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ANNALS
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VOL. 67 MARCH, 1958 No. 1

I

ARTERIAL OBSTRUCTION OF THE LABYRINTH
PART I. COCHLEAR CHANGES

ROBERT KIMURA, B.A.
AND
H. B. PERLMAN, M.D.
CHICAGO, ILL.

Our recent experiments[1,2] have shown that acute obstruction of the inferior cochlear vein and its tributaries produces severe functional and histological changes of the auditory and vestibular end organs. An orderly sequence of cellular damage was established by studying lesions at frequent intervals after obstruction. Similar information on the effect of acute interruption of the arterial flow to the inner ear is needed. Perusal of the literature reveals that no systematic study of arterial obstruction to the labyrinth has been carried out. Earlier investigators (i.e. Knick,[3] Wittmaack,[4] Kaida,[5] Hosomi,[6] Hirose[7] and Borghesan[8]) who reported histological changes were concerned primarily with the problem of end organ changes following injury to the cochlear nerve trunk. Their blind surgical approach towards the internal meatus and the simultaneous surgical damage to the VIII nerve trunk made it difficult to interpret the histological changes. Severe degeneration of the membranous labyrinth occasionally seen in their experiments was attributed to accidental damage to the internal auditory artery. A six day and a thirty day lesion of this kind were

From the Division of Otolaryngology of the University of Chicago.

This work was aided in part by funds from U.S. Public Health Grant B269 (C3) Douglas Smith Foundation for Medical Research of the University of Chicago and Central Bureau of Research of the American Otological Society. Also by the U. S. Air Force contract No. AF 41(657)143 monitored by the School of Aviation Medicine, USAF, Randolph Air Force Base, Texas.

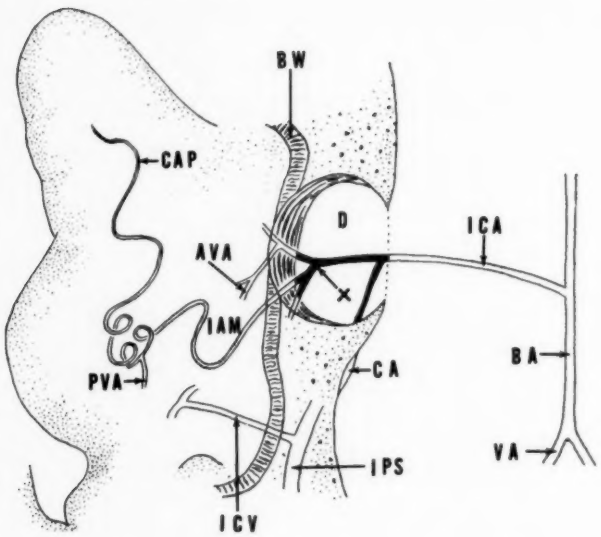

Fig. 1.—Schematic drawing showing approach for surgical interruption of the labyrinth arteries. VA—Vertebral artery; BA—Basilar artery; ICA—Anterior inferior cerebellar artery; AVA—Anterior vestibular artery; PVA—Posterior vestibular artery; CAP—Cochlear artery; ICV—Inferior cochlear vein; IPS—Inferior petrosal sinus; CA—Cochlear aqueduct; BW—Bullar wall; D—Surgical defect; IAM—Internal auditory meatus; X—Point of arterial obstruction.

described by Knick. In more recent studies concerned with electrical and conditioned responses of the cochlea after nerve section, the effect of associated damage to the arterial supply was occasionally noted. At the same time the finer histological changes due to vascular lesion in the human labyrinth have been difficult to assess due to post-mortem degeneration.

The effect of a pure vascular lesion without simultaneous damage to the VIII nerve has not been clear. Such a lesion is difficult to produce. Examination of human material by Hopmann[9] in 1904 and our examination of the guinea pig with latex and with lead chromate injections indicate the wide variations in branching and collateral anastomosis in this area. Nevertheless sufficient regularity in the location of these arterial divisions of the anterior inferior cerebellar artery and their surgical accessibility a few millimeters away from the VIII nerve trunk proper was established.

The purpose of this report is to present in some detail the histopathological changes which occur at various time intervals after arterial obstruction. A preliminary report comparing some of these histological changes and those found after venous obstruction was recently made.

MATERIALS AND METHODS

A total of 65 guinea pigs weighing from 250 to 450 grams were used.

The surgical approach to the anterior inferior cerebellar artery was ventral through the bulla cavity. The area of drilling on the petrous bone was directly medial to the basal turn, anterior to the intracranial opening of the cochlear aqueduct and of the inferior cochlear vein (Fig. 1). While drilling, the inferior petrosal sinus and the brain were protected by a very small piece of the non-toxic and flexible plastic placed against the dura. The drilling was stopped at approximately two millimeters above and outside of the internal auditory meatus. In this bony defect, the labyrinthine branches of the anterior inferior cerebellar artery can be seen through the dura mater. After the dura was incised, the arteries were lifted and coagulated simultaneously with a small suction tip. The suction tip was made from a surgical syringe needle Number 27 insulated except at the tip. The defect in the otic capsule was filled in with a piece of gelfoam, but the opening on the bulla was covered with only the overlying soft tissue. The tympanic membrane was not damaged.

Thus the labyrinthine arteries were obstructed at the outside rather than inside the internal auditory meatus. With this procedure the cochlear nerve trunk, inferior cochlear vein and cochlear aqueduct were not damaged. Variations in branching made for some differences in the vessels surgically accessible to coagulation.

In the preliminary phase of the experiment, a fenestra was made in the apex of the cochlea to observe the change in the vascular flow resulting from obstructing the arteries at this point. It was found that the blood flow could be completely stopped. We were reasonably certain that in the many subsequent operations even though the circulation was not checked at the apex, the chance of interrupting the labyrinthine circulation was high.

Animals were sacrificed at different intervals; one-half, one, two, three, four, five, six, sixteen and seventeen hours, one, two, three, four,

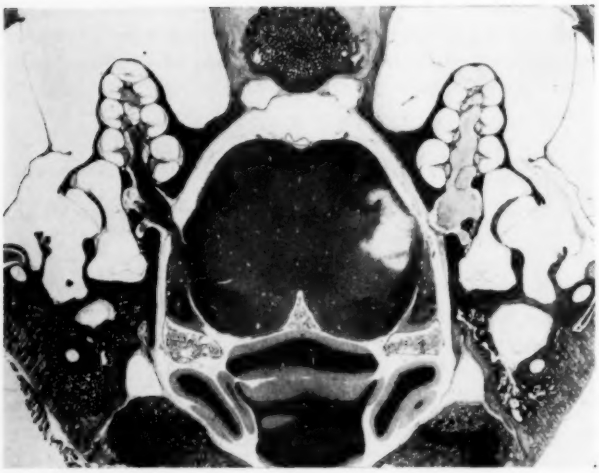

Fig. 2.—Section through both labyrinths and brain stem showing operated and unoperated sides with extensive lesion after 24 hours.

five, six, seven, eight, nine, ten, twelve, fourteen, twenty-one days, one, two, three, four, five and six months; two or more animals in each group. As reported in our experiments on venous obstruction only one ear was operated upon, while the other ear served as a control. Five animals were operated upon in the same manner but without coagulating the arteries. These showed no labyrinth pathology.

All the animals were perfused with Heidenhain-Susa fixative. Both ears were kept intact with mid brain and cerebellum, and were processed with the celloidin method. The specimens were cut horizontally at 18 microns so that the histological details of both cochleae could be compared in the same region. Every fifth or tenth section was stained with H and E or Mallory's connective tissue stain (Fig. 2).

FINDINGS

Symptoms. All animals successfully operated upon showed vestibular disturbances on awakening but this varied in intensity and duration. Some animals recovered in a few days and others showed signs of vestibular imbalance throughout the period of observation. Nystagmus usually disappeared after one or two days while disturbances in posture and gait were more lasting. Caloric tests were carried

out on a number of the chronic animals. Reduced or complete absence of vestibular response even to ice water on the operated side could be demonstrated.

Cochlear Circulation. In a number of the acute animals and in some of the chronic preparations at the time of sacrifice a fenestra was made in the apical turn to observe the blood flow. In the acute stage complete cessation of blood flow was seen in the vessels of the exposed spiral ligament and stria while the vessels remained filled with blood. In the chronic animals the fenestrated area was devoid of blood vessels.

Histopathology. Variation in branching of the terminal labyrinth vessels with respect to the arterial trunks exposed in the surgical field made for differences in the resulting damage. Almost all animals in which coagulation of an artery was carried out showed some histological changes in the labyrinth. Forty-two per cent showed extensive degeneration of vestibular and cochlear end organs. Thirty per cent showed severe lesions limited to the cochlea, saccule and posterior ampulla. In two animals only the utricle, horizontal and superior ampulla and part of saccule were affected. These are supplied by the anterior vestibular artery. In almost twenty per cent of the animals there was no symptomatic evidence of arterial obstruction of the labyrinth despite coagulation of an exposed arterial trunk. However, in this group scattered degeneration of the sensory cells near the round window was noted.

Hair Cells. The hair cells of the organ of Corti were strikingly vulnerable to this lesion. A progressive series of histological changes can be established (Fig. 3).

The nuclei of the inner hair cells became pyknotic and their cytoplasm edematous within a half hour. Within three hours there is complete disintegration of the inner hair cells in some areas while the outer hair cells are still discernible. Within four hours both inner and outer hair cells are widely disintegrating. At six hours most of the hair cells have disappeared. These changes occurred throughout the cochlea with the exception of the area immediately adjacent to the round window. This portion of the organ of Corti, about 5 per cent of its total length, changed more slowly and showed more vulnerability of outer hair cells.

Supporting Cells of the Organ of Corti. The pillar cells are definitely affected by this lesion. Disintegration of the inner pillar at

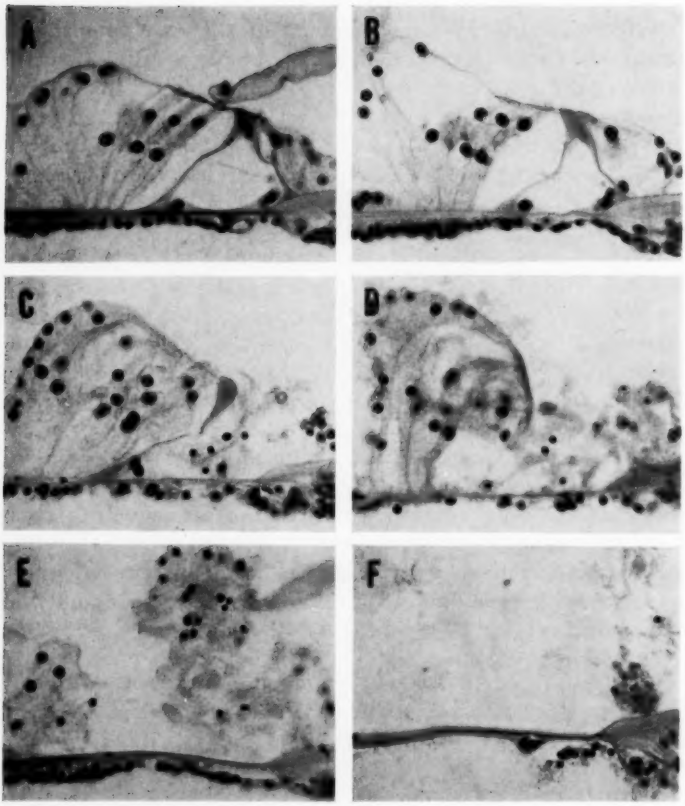

Fig. 3.—Progressive changes of the sensory and supporting cells of the organ of Corti after arterial obstruction. A. Normal. B. Inner hair cell swelling at one hour. C. Disintegration of inner hair cells and inner pillar at three hours. D. Collapse of the organ of Corti with loss of pillars at four hours. E. Disintegration of the whole end organ at six hours. F. Denudation of basilar membrane at 17 hours.

three hours followed by the outer pillar and collapse of the reticular plate were consistent findings throughout the cochlea except for the round window area.

Within an hour after the pillar cells disintegrate Deiters and Hensen cells begin to separate from the basilar membrane and undergo similar changes. The external sulcus cells show edema and pyknosis within an hour and soon disappear along with the other supporting cells. The cells of Claudius and internal sulcus cells are slightly more resistant but at six hours also begin to separate from the basilar membrane. Sixteen hours after arterial obstruction all the supporting cells except for the Boettcher cells have completely disappeared from the basilar membrane and appear as cellular fragments in the cochlear duct. The Boettcher cells may persist for a few more days.

Basilar Membrane. The basilar membrane remains intact throughout the period of extensive disintegration of the end organ. It becomes slightly thicker than normal. The mesothelial cells directly below the basilar membrane show pyknosis and reduction in number by four hours. They disappear by 17 hours. The blood vessels beneath the tunnel of Corti contain blood cells and appear to course within the fibers of the basilar membrane. After two weeks, with the onset of fibrosis and ossification of the cochlea, the basilar membrane finally disappeared.

Tectorial Membrane. The tectorial membrane becomes detached from the spiral limbus in three hours to be free in the cochlear duct. Compared with the control side it is definitely swollen. The process of disintegration is very slow. It can still be identified after fibrosis and ossification has started (Figs. 7 and 8).

Stria Vascularis (Fig. 4). The stria was particularly vulnerable. It began to detach from the spiral ligament close to the spiral prominence within an hour. At the same time globules appeared on the surface of the stria. No definite swelling or hemorrhage was seen. Within four hours the entire stria had separated from the spiral ligament and was beginning to disintegrate. As in the case of the other elements of the cochlear duct the stria in the region adjacent to the round window changed more slowly.

Spiral Ligament (Fig. 4). Within one hour there was pyknosis of cells near the external sulcus and also adjacent to the endosteum. At this time small globules were seen on the surface of the spiral liga-

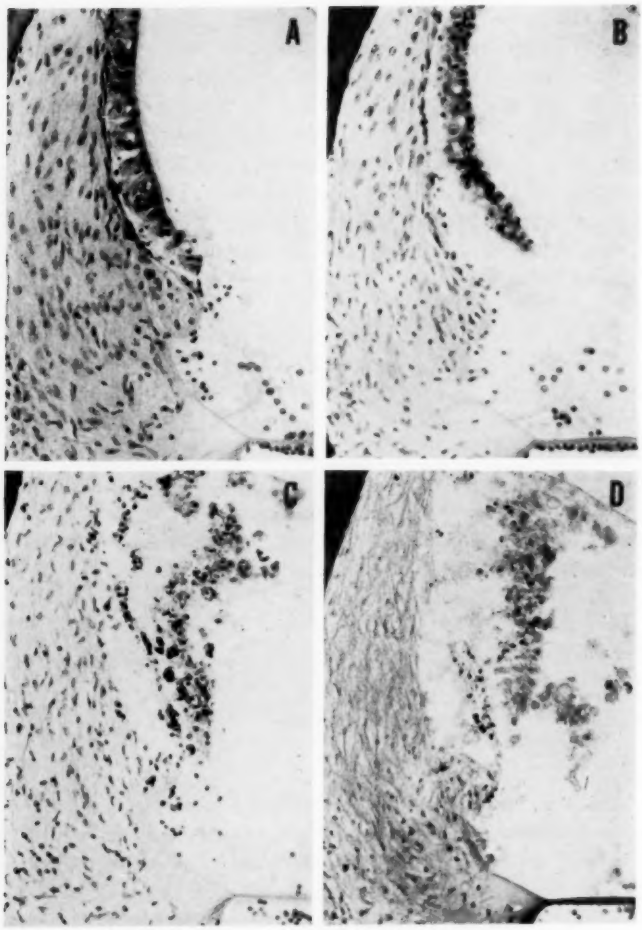

Fig. 4.—Progressive changes in the stria vascularis and spiral ligament after arterial obstruction. A. Beginning detachment starting at the spiral prominence at three hours. B. More advanced separation at four hours. C. Advanced separation and disintegration at one day. D. Fragments still visible at 12 days.

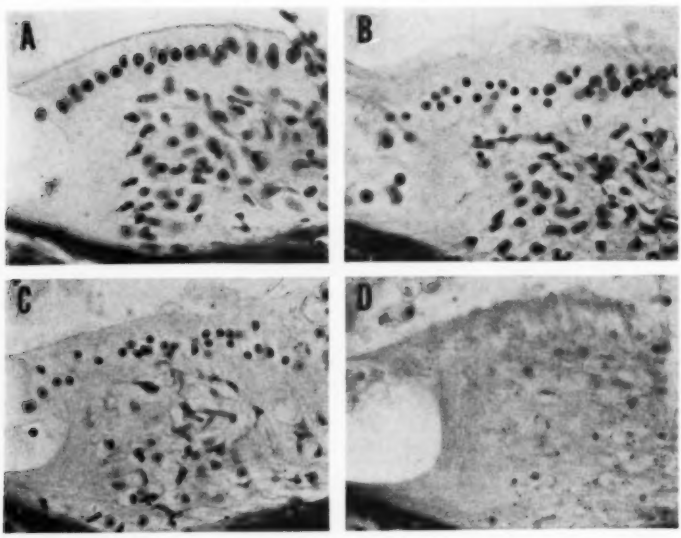

Fig. 5.—Progressive changes in limbus spiralis following arterial obstruction. A. Normal. B. Three hours after obstruction. C. Four hours after obstruction. D. Fourteen days after obstruction—when almost all the cells have disappeared.

ment on the scala vestibuli side adjacent to the attachment of Reissner's membrane. Pyknosis and detachment of the epithelial cells of the spiral prominence was also definite at one hour. After six hours most of the cells became involved. Then they fragmented and disappeared with little or no signs of phagocytosis. The supporting acellular fiber structure of the spiral ligament remained. After two weeks this was invaded by cells from the endosteal layer and ossification started in one month. The entire spiral ligament became ossified by five months but its shape could still be identified. Hemorrhage or edema was not seen at any time.

Limbus Spiralis (Fig. 5). The first changes were seen at three hours when the cells near the vestibular lip became pyknotic. All the cells rapidly became involved throughout the cochlear duct and disappeared by two days leaving an acellular fiber system that began to ossify in one month. Its shape remained discernible even when completely ossified.

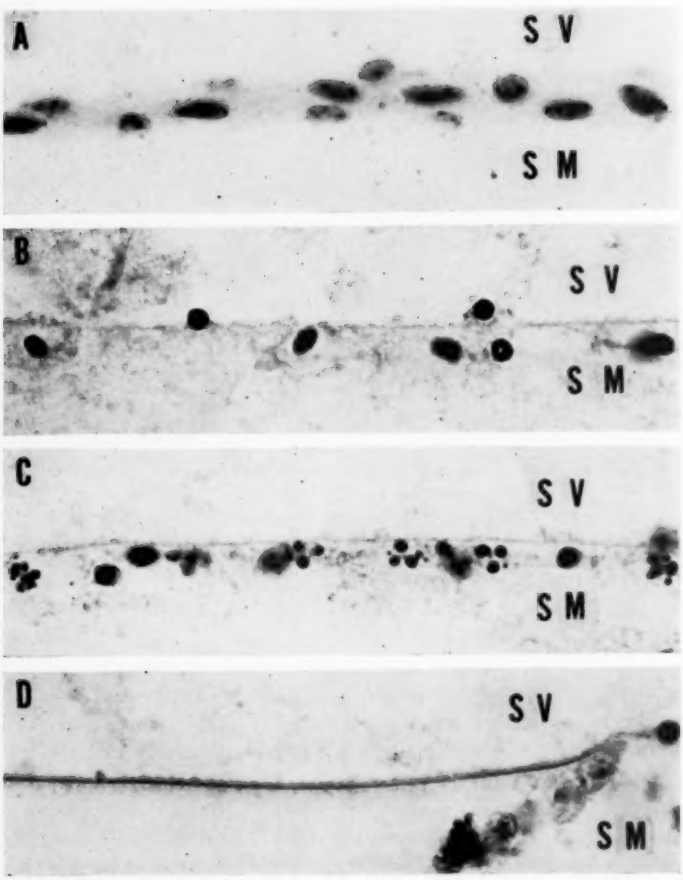

Fig. 6.—Progressive changes in Reissner's membrane starting with normal A, 17 hours after arterial obstruction B; at 24 hours C; six day lesion D when both mesothelial and endothelial cells have disintegrated leaving a homogeneous basement membrane. S.V. Scala vestibuli; S.M. Scala media.

Reissner's Membrane (Fig. 6). The cells on both sides of this membrane became pyknotic in four hours, fragmented in twenty-four hours and finally disappeared leaving an amorphous basement membrane by the sixth day. The cells along the entire width and length were affected simultaneously except for the area adjacent to the round window. Changes in this area were somewhat delayed. Reissner's membrane did not disintegrate or rupture even when acellular. It was replaced by fibrous tissue at two weeks. Neither bulging or collapse of Reissner's membrane was seen at any time.

Cochlear Duct (Figs. 7 and 8). The cellular fragments from the organ of Corti and stria vascularis remained free for days in the scala media without complete dissolution and without aggregating in any particular location. Hemorrhage was strikingly absent. Free phagocytes were rarely seen. The free space of the scala media was reduced and obliterated by fibroblasts. This began at the basal turn eight days after arterial obstruction and was complete here at one month. Then calcification of this tissue set in and proceeded to complete ossification within six months. These changes spread toward the apex from the basal turn.

Perilymphatic Space. No hemorrhage or other cellular elements were observed in the perilymphatic spaces after arterial obstruction. Fibrosis and ossification proceeded at about the same rate and direction as in the scala media. At the end of six months the entire cochlea was filled by intramembranous ossification (Fig. 9).

Otic Capsule. The otic capsule did not demonstrate any immediate change. It could be identified even when the entire cochlea was filled with new bone. In a few chronic specimens with ossification the upper turns down to the second turn had disappeared, apparently absorbed, while the lowest turns were filled with new bone. The footplate of the stapes and the capsule bone around the oval window also showed no indication of abnormal activity even though fibrosis and ossification was present in the vestibule. New bone is laid down against that portion of the otic capsule that forms the walls of the modiolus. It finally invades and replaces the old bone with marked narrowing of the spaces occupied by the cochlear nerve trunks.

Blood Vessels. The histological changes in the vessels are progressive. The blood cells undergo lysis and are replaced by hyaline material. The arteries become narrowed while the caliber of the posterior spiral vein remains unchanged. Ultimately the vessel wall dis-

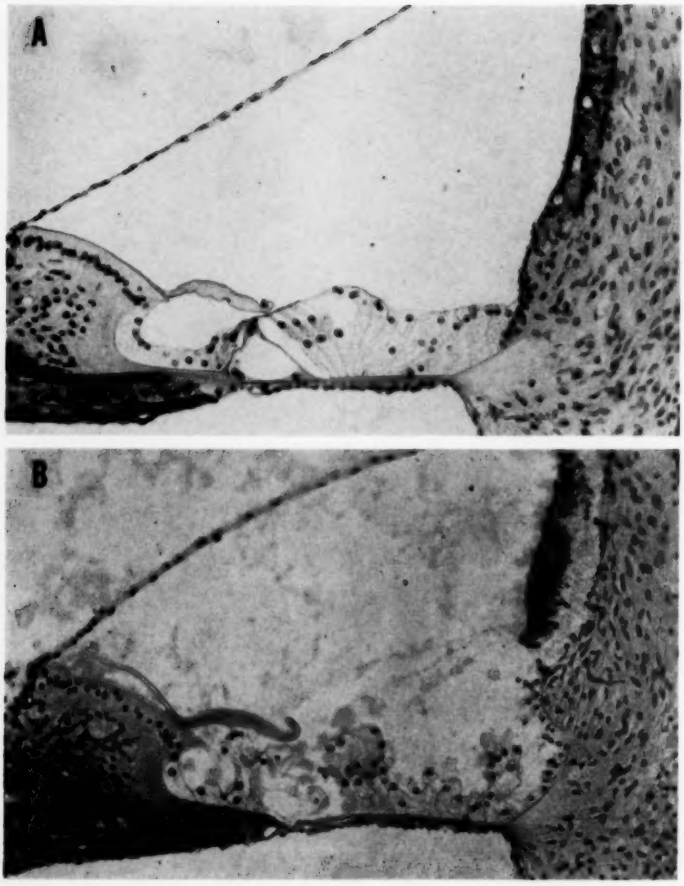

Fig. 7.—Changes in the cochlear duct four hours after arterial obstruction (B). Note separation of the stria and the tectorial mambrane; disintegration of the pillars and inner hair cells. A is normal.

appears and their bony channels are invaded by new bone. As growing fibrous tissue obliterates the fluid spaces new thin-walled blood vessels appear. The vessels around the endolymphatic sac were consistently empty indicating free blood flow. They did not proliferate. Vascular anastomosis between the middle ear and the labyrinth were rarely seen.

Spiral Ganglion (Fig. 10). The first visible change in the spiral ganglion is the reduction of the tigroid substance in the cytoplasm within one hour followed by chromatolysis of the nucleus in two hours. A few hours later the ganglion cell nuclei are pyknotic and the cytoplasm shrunken. These ganglion cells persist for many weeks until replaced by fibrous tissue. Loss of ganglion cell began eight days after obstruction and was complete in two months. Myelophages were evident in the fourteen day lesion along with proliferation of fibroblasts. Ultimately these areas became ossified. The entire length of the spiral ganglion was affected by this lesion. However, the ganglion cells disappeared first in the basal turn. This dissolution of ganglion cells spread slowly towards the apex along with the progressive fibrosis of the cochlea. The fibers of Rasmussen's bundle also degenerated.

Cochlear Nerve and Nuclei. The entire cochlear nerve trunk stained poorly with eosin one hour after arterial obstruction. Pyknosis of the Schwann cell nuclei was evident at four hours. Myelin droplets appeared in four days at the level of the lower turns and progressed to the apex. Myelophages then appeared in the same order. The ventral cochlear nucleus was often involved. The dorsal cochlear nucleus was rarely involved by this lesion. The cells of the ventral cochlear nucleus were affected as early as those of the cochlear nerve trunk and spiral ganglion.

Blood vessels in this region were filled with blood indicating obstruction of the circulation. In the chronic lesions of the cochlear nucleus there was a greater reduction in the nerve fibers than in the nerve cells. The total volume of the nucleus was reduced to about half normal. The blood supply to some of the adjacent nervous tissue was also interrupted and localized necrosis of nervous tissue in the brain stem resulted. The restiform body, cells and tract of the V nerve and occasionally the cells of the vestibular nuclei and olive were affected.

COMMENT

It is possible to interrupt the arterial supply to the inner ear without trauma to the VIII nerve trunks. This produces a pure vascular

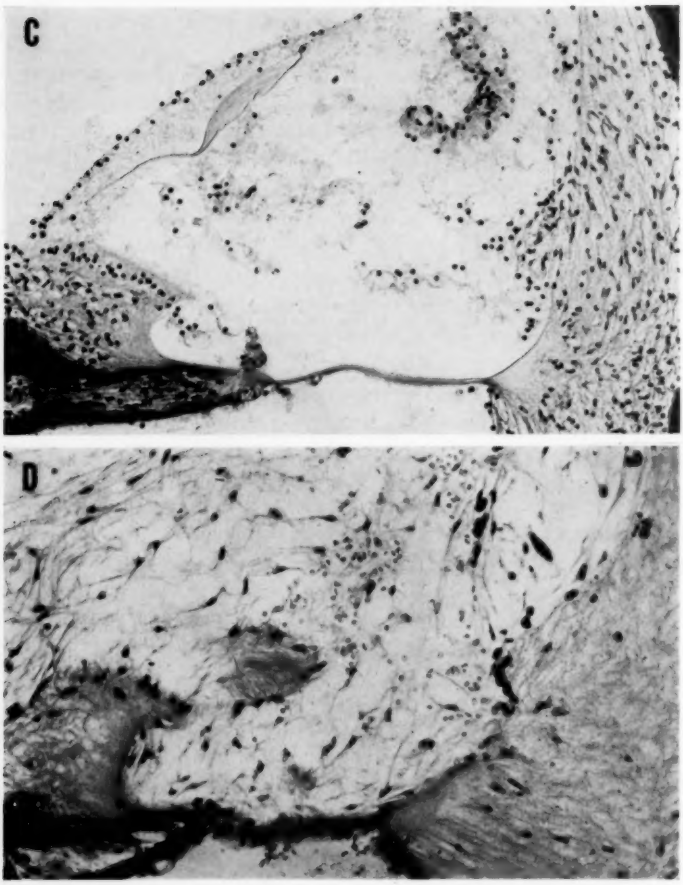

Fig. 8.—More advanced changes in the cochlear duct after arterial obstruction. C. 17 hour lesion showing dissolution of end organ and stria. D. Two week lesion with fibrous tissue filling cochlear duct.

lesion that is not complicated by simultaneous surgical damage to the nerves. This experimental arterial occlusion produces a severe localized brain stem lesion. This lesion resembles that seen in man following cerebellar artery thrombosis and suggests the possibility that such disease in man may also involve the labyrinth. Examination of temporal bone sections from such cases may clarify this point.

Profound and rapid changes take place in all the structures of the cochlea. An orderly sequence of histological events can be established beginning with changes of hair cells in half an hour followed in a few hours by involvement of the supporting cells and finally to complete ossification of the cochlea in six months. The vulnerability of the hair cells, pillar cells and the stria is noteworthy. Necrosis of the structures followed by fibrous tissue ingrowth and ossification is not unlike that observed in chemical or bacterial labyrinthitis. However, infection did not play a part in the pathological changes of the labyrinth produced in our experiment although evidence of limited middle ear infection was occasionally encountered. Simultaneous changes in the end organ, spiral ganglion, nerve trunk and acoustic nuclei indicated the common factor of interference with the blood supply to all these areas. Other investigators concerned with the problem of VIII nerve section have described extensive cellular changes in the cochlea resembling some of the older lesions in our series. These were undoubtedly the result of accidental damage to the arterial supply. The reason for the simultaneous degeneration of the efferent nerve bundle of Rasmussen is not clear but the histological changes are definite.

The early changes following arterial obstruction closely resemble post-mortem degeneration of the cochlea.[10,11] The former are more rapid and severe. They are followed by the reparative process of fibrosis and ossification beginning about eight days after operation and ending in complete ossification with occasional resorption of the ossified apical portion.

Arterial obstruction causes much more rapid and severe damage to the cochlea than venous obstruction. Changes in the stria and organ of Corti in a few hours after arterial obstruction are more severe and widespread than those after weeks of venous obstruction.

Observation of the blood flow in the acute and chronic animals with arterial obstruction indicates complete cessation of all blood flow in the exposed stria and spiral ligament. In contrast similar observations after venous obstruction indicate marked slowing but not com-

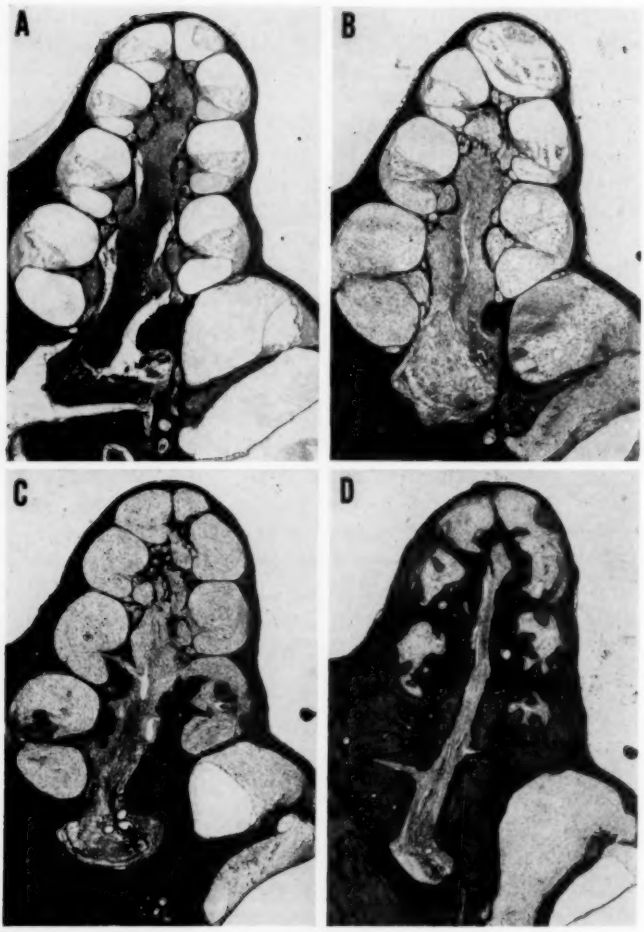

Fig. 9.—Progressive fibrosis and ossification from base to apex of cochlea. A. Before fibrosis begins—17 hour lesion. B. One month after arterial obstruction. C. Two months after arterial obstruction. D. Five months after arterial obstruction.

plete cessation of flow except in some of the strial vessels where blood flow may stop. This difference in the effects on the cochlear circulation may in part account for the difference in degree and rapidity of histological changes. Fibrosis and ossification did not occur in the cochlea even after five months of venous obstruction. Hemorrhage in the endolymphatic and perilymphatic spaces uniformly following venous obstruction was strikingly absent after arterial obstruction. After arterial obstruction the stria vascularis appeared to lift away from the spiral ligament as one unit, without edema, hemorrhage or dilation of vessels. After venous obstruction the stria did not lift away but edema, hemorrhage, vessel dilation and fragmentation was produced. The stria finally disappeared and was replaced by a low flat epithelium. Arterial obstruction first produced necrosis of the inner hair cell and inner pillar in contrast to venous obstruction which rarely affected the pillars and damaged the outer hair cells first. The explanation for this difference is not apparent. It is interesting that Hawkins and Lurie[12] found the inner hair cell to be more susceptible to streptomycin. In arterial obstruction the supporting cells rapidly become detached from the basilar membrane while in venous obstruction these cells remain in place as they degenerate. In both arterial and venous obstruction the damage along the cochlear duct was widespread but after arterial obstruction it was continuous rather than scattered. Complete denudation of the basilar membrane and of Reissner's membrane was a striking feature of the arterial lesion not seen following severe venous obstruction. Swelling of the tectorial membrane and detachment from the spiral limbus was also unique to arterial obstruction. Rapid severe widespread changes in the spiral ganglion and nerve trunk were a primary effect of interruption of their blood supply while the limited lesions in the spiral ganglion and nerve trunks seen much later after venous obstruction were probably the result of secondary degeneration following end organ damage.

The spiral ligament degenerated uniformly after arterial obstruction while after venous obstruction degeneration was limited to the region adjacent to the external sulcus. Spiral limbus degeneration including the cells of Huschke's teeth is severe and widespread after arterial obstruction while after venous obstruction this area shows only limited small lesions. Similar to venous obstruction, there was neither collapse or bulging of the Reissner's membrane after arterial obstruction. However, there was almost complete degeneration of the two layers of cells comprising this membrane. This left a homogeneous basement membrane which persisted in a normal position and was finally replaced by fibrous tissue.

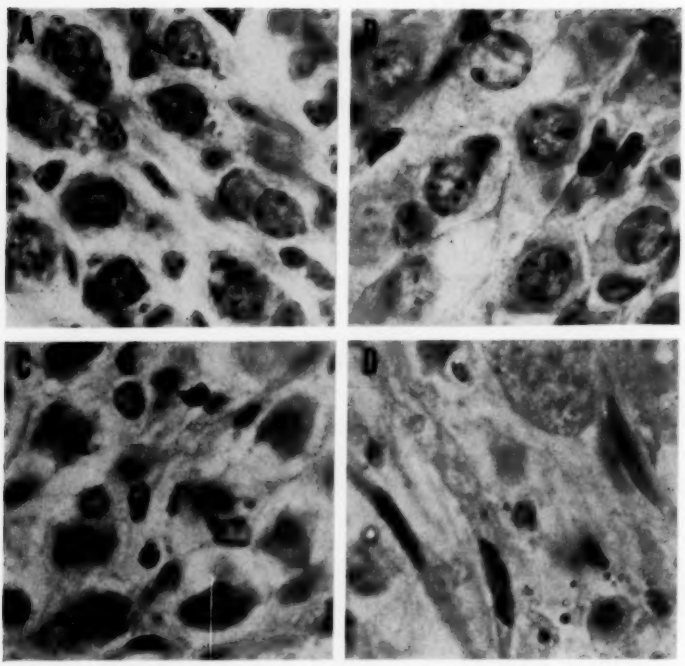

Fig. 10.—Changes in spiral ganglion cells. A. Normal. B. Reduction of Nissl substance and chromatolysis in two hours. C. Pyknosis at four hours. D. Dissolution of ganglion cells at 14 days.

The presence of blood-filled vessels was a good indication of a successful arterial obstruction since all animals were fixed by intravital perfusion.

The sequence and character of the pathological changes produced by acute arterial obstruction of the labyrinth vessels are well defined in this experiment and should be a guide to the interpretation of human temporal bone pathology with regard to this cause for sudden deafness. Differences in the exact terminal branches exposed and coagulated account for variation in the degree and extent of pathology seen in these animals. Moreover, collateral circulation beyond the point of coagulation may account for only partial or temporary impairment of blood flow in some. The retraction of the inferior petrosal sinus

and its attached inferior cochlear vein in the control operations was not followed by visible pathology. Similar to the findings after venous obstruction cellular fragments of the stria and end organ appeared to disintegrate in the cochlear duct without phagocytosis or movement along the duct.

SUMMARY

Surgical obstruction of the arterial supply to the cochlea produces rapid widespread and profound damage. There were simultaneous changes of ganglion cells, nerve, end organs and supporting tissues. Reparative processes began in eight days and were characterized by fibrosis and ultimate ossification of the cochlea. The earlier changes closely resemble that seen in post mortem degeneration. An orderly sequence of pathology was established from one-half hour to six months after arterial obstruction and shows important differences from those previously reported after extensive venous obstruction. On the basis of the pathological changes reported here, the relation between arterial occlusion and sudden loss of hearing may be clarified in human temporal bone sections.

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ARTERIAL OBSTRUCTION OF THE LABYRINTH
PART II. VESTIBULAR CHANGES

There is little information about the histopathology of the vestibular apparatus associated with vascular lesions. Lindsay and Hemenway[1] recently described a case of sudden vertigo and reduced vestibular function with no auditory symptoms. A vascular anomaly (marked dilation and hypertrophy of vessels in the internal auditory meatus) was found associated with degeneration of the utricular nerve and the nerves to horizontal and superior canal ampullae. Experimental results following extensive venous obstruction of the labyrinth have been reported by us recently. The effect of acute arterial obstruction of the vessels going to the vestibular labyrinth has not been systematically studied. Arterial obstruction of the labyrinth vessels was experimentally produced without surgical trauma to the VIII nerve trunk. The resultant cochlear changes are described in Part I. Systematic changes in the vestibular portion of the labyrinth following acute surgical interruption of the arterial supply are presented here. The manner of branching of the anterior-inferior cerebellar artery at the point of sugical exposure suggests the possibility of interruption of blood flow limited to the vestibular portion of the labyrinth. A clinical example may be sudden loss of vestibular function without hearing loss—occasionally seen in patients.

Material and Method. Operations were performed on 65 guinea pigs. The surgical exposure and techniques have been described in the previous report on the cochlea. The vestibular end organs are supplied by two arteries. One, the anterior vestibular, is often a direct branch of the anterior inferior cerebellar artery and supplies the utricle, the superior and horizontal canal ampullae. The other (posterior vestibular artery) is a branch of the common cochlear artery supplying the saccule and posterior canal ampulla. The manner of branching and the exposure obtained did not permit interruption of the anterior vestibular artery alone. Study of injected material indicated that collaterals exist beyond the point of coagulation that would favor the maintenance of blood flow in the anterior vestibular artery. Careful postoperative observations as to vestibular signs were made up to six months following surgery. In a number of animals caloric tests were carried out.

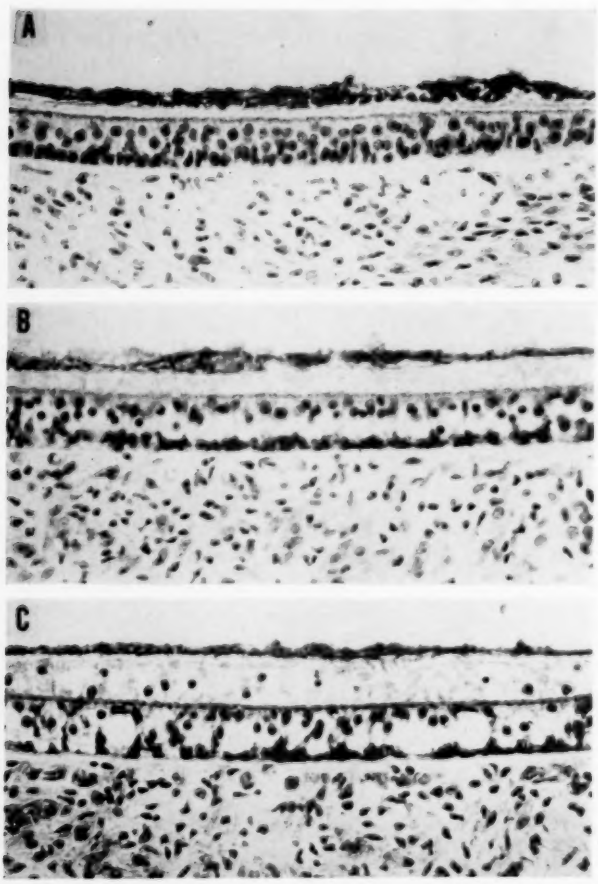

Fig. 1.—Sections through the saccular macula showing effect of acute arterial obstruction. A. Normal control. B. Separation of sensory cell from the supporting cells and pyknosis after one hour. C. Further separation of cells, elevation of otolith and cells in subotolith space at two hours.

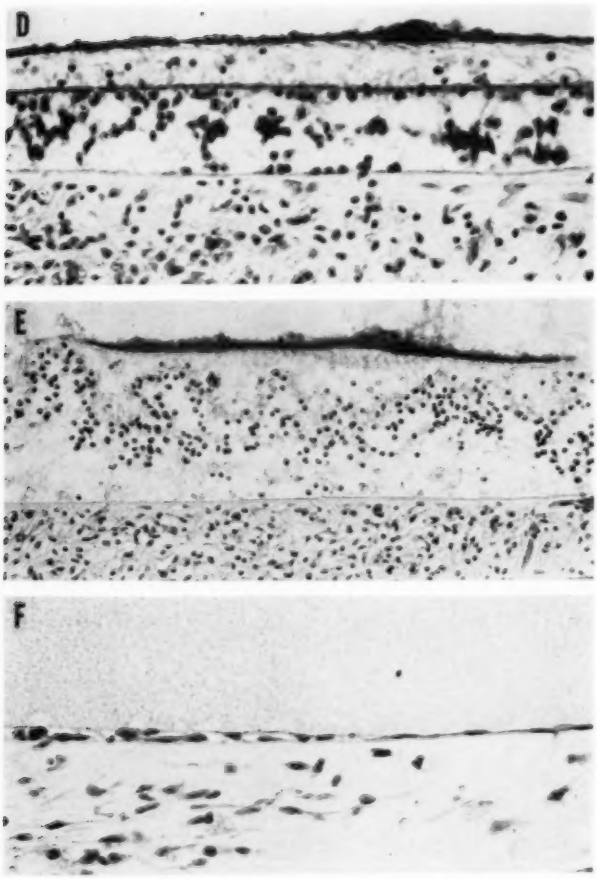

Fig. 2.—Further changes in the saccular macula after arterial obstruction. D. Disintegration of sensory and supporting cells at six hours. E. Further disintegration with elevation and retention of otolith at 17 hours. F. Disappearance of macula at 14 days.

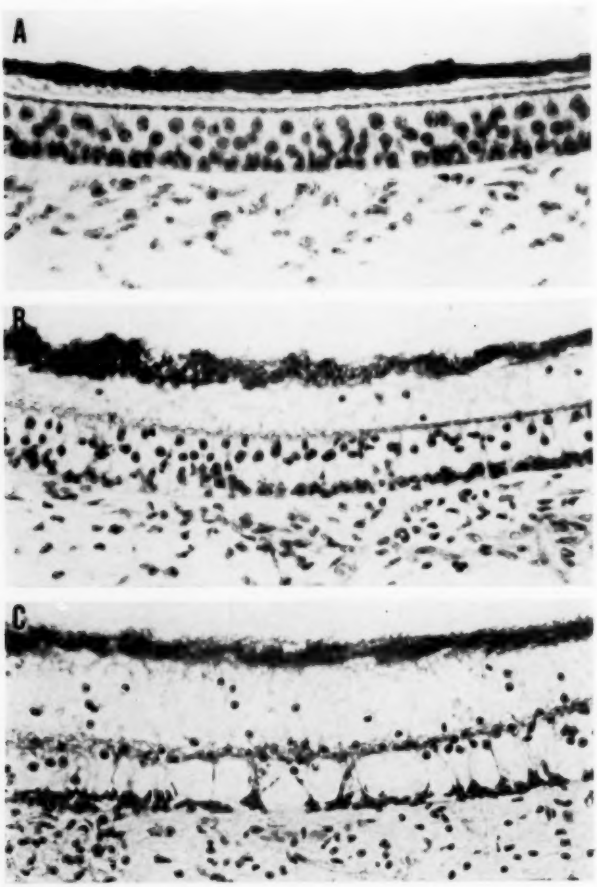

Fig. 3.—Section through the utricular macula showing changes due to arterial obstruction. A. Normal control. B. Edema, pyknosis and elevation of otolith at three hours. C. More advanced changes at 16 hours.

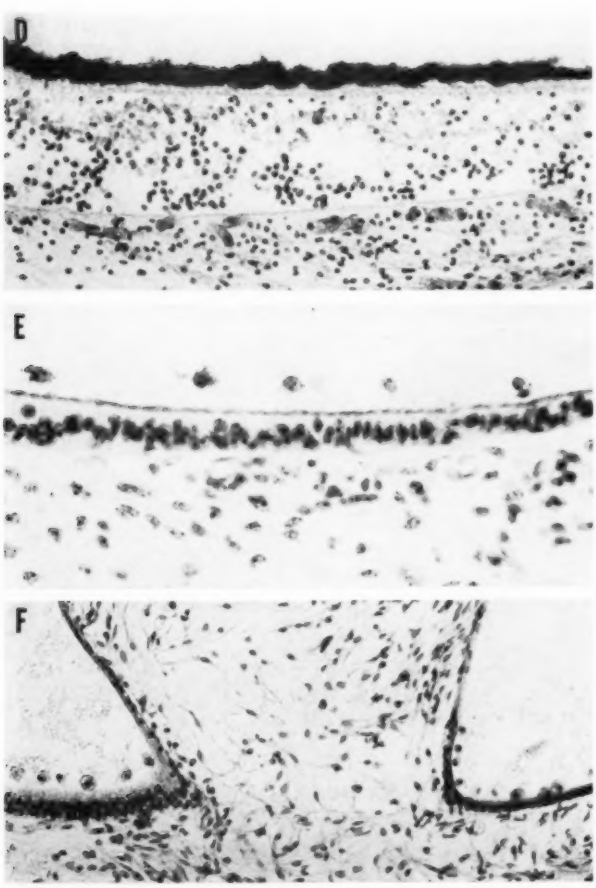

Fig. 4.—Further changes in the utricular macula. D. Advanced cellular disintegration with preservation of otolith at 24 hours. E. Disappearance of sensory cells with retention of supporting cell fourteen days after arterial obstruction. F. Fibrosis in atrophic macular area at 14 days indicating reparative processes after more severe necrosis.

The exposure of the posterior fossa of the skull and severe post-operative reactions accounted for a mortality of about 17 per cent as compared to a mortality rate of about 5 per cent in the venous obstruction experiments.

Vestibular Signs. Severe labyrinth symptoms were usually seen after this procedure. Nystagmus, body and neck torsion, and rolling were more marked than after acute venous obstruction. The simultaneous effect of this arterial obstruction in the adjacent vestibular and other brain stem nuclei would undoubtedly explain some of the more severe and lasting signs seen in these animals. While nystagmus disappeared in a few days, ataxia and inability to stand and move were sometimes very pronounced for one week. However, stumbling and falling toward the operated side was seen in animals many weeks after operation. Ultimately their gait became more normal.

When the spontaneous nystagmus had disappeared caloric tests were carried out in a number of animals and all degrees of response were noted from total absence with ice water in the operated side to a normal response with water at 20° C.

Histological Findings. As in the cochlea, profound and extensive damage to the vestibular end organs can be produced by experimental arterial obstruction. However, the changes are not as advanced as in the cochlear portion. In about 20 per cent of the animals no vestibular lesions were found on histological examination. The remainder showed various degrees of damage. The findings in each end organ will be detailed.

Saccule (Figs. 1 and 2). Seventy-two per cent of all the operated animals show lesions of the saccule. In more than half of these the entire labyrinth is involved. In a little less than half the lesion is limited to the cochlea, saccule and posterior ampulla. In these animals the superior part of the saccular macula is usually spared.

As in the organ of Corti the sensory cells are most vulnerable. Changes were visible one hour after arterial obstruction. There was pyknosis of hair cell nuclei with edema. Separation of the hair cell layer from the supporting cells was seen within two hours along with migration of hair cell nuclei into the subotolith space. As the neuro-epithelial layer swells the cuticular plates disintegrate and at seventeen hours a mass of necrotic cells occupies the space between the elevated otolith and the denuded basement membrane. The supporting cells and the subepithelial connective tissue become pyknotic and begin to

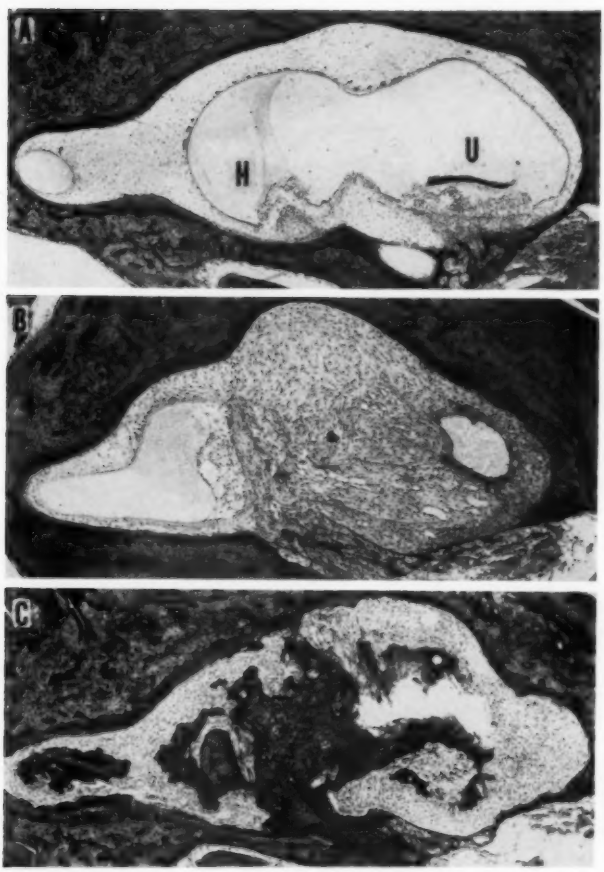

Fig. 5.—Sections through entire utricle and horizontal canal ampulla following arterial obstruction. A. Dissolution of macula and crista at 17 hours. B. Reparative process of fibrosis in the same area at one month. C. Further reparative process with extensive ossification at three months.

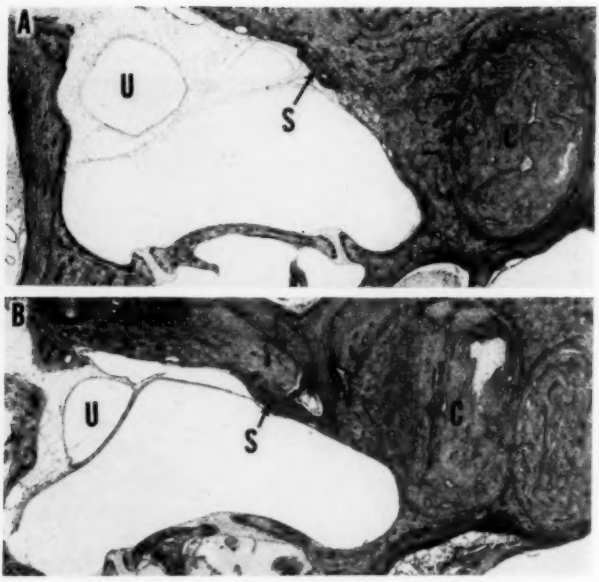

Fig. 6.—Section through the vestibule and cochlea showing that the cochlea may be completely obliterated by bone while the vestibule remains remarkably free of either fibrosis or ossification. A. Five months following arterial obstruction. B. Two months following arterial obstruction. U. Utricle. S. Ossified saccular macula. C. Bone entirely fills the cochlea.

disintegrate soon after the hair cells. Changes in the saccular nerve occur at about the same time. In one week all cells and the otolith disappear and then a single layer of cells replaces the original end organ. This is followed in another week by fibrosis and in eight weeks by ossification of the macular area.

The saccular wall is more resistant but shows pyknosis in six hours. The cells are reduced in number and some are replaced by a cuboidal cell in six days. Neither collapse nor dilation of the saccule was observed. The saccule gradually fills with fibrous tissue and bone in six months. Blood cells were rarely seen in the saccule. The cellular debris did not appear to migrate.

While usually the inferior part of the macula was involved, occasionally only the superior part of the macula was affected. Here the sensory cell was lost while the supporting cell remained.

Utricle (Figs. 3 and 4). The utricle showed lesions in 50 per cent of all operated animals. Again the sensory cells showed the first signs of damage. The type and rate of pathology resembled that seen in the saccular macula except that the supporting cells were occasionally unaffected. Fibrosis and ossification were more rapid and extensive in and around the utricle than in the saccule and vestibule (Figs. 5 and 6).

The position of the wall of the utricle was not affected but the epithelial cells were damaged as in the saccule and in addition the pigment cells normally found here underwent change. Rounding of cells, concentration of pigment granules, disintegration of cells with freeing of pigment and displacement of the cell from the wall into the perilymphatic meshwork and endolymph were noted.

The cells of the utricular wall adjacent to the pigment cells became detached from the basement membrane in three days. Cellular fragments were most prominent in the region of the macula where they appeared to disintegrate. The region about the utriculo-endolymphatic valve was free of cellular material. Blood cells were rarely seen in the utricle or about it.

Ampullae and Canals (Fig. 7). The ampulla of the posterior canal was more often involved than the horizontal and the superior because of the particular vessel coagulated. The latter canal lesions were always associated with utricular damage. Again the sensory cells of the cristae showed the greatest vulnerability. Within an hour after arterial obstruction, edema and pyknosis were evident in these cells. This rapidly involved the supporting cells and within one day the entire crista began to separate from the mound of connective tissue cells. Ossification of this area was seen at two months.

The cells of the planum semilunatum were somewhat more resistant but within a few days they became separated from the basement membrane.

Unlike the control side the cupular substance was not shrunken but appeared unusually well preserved. The cupular substance persisted until replaced by fibroblasts. The wall of the ampulla and the rest of the membranous canal undergo changes similar to that of the utricle. Within two weeks fibrosis begins and by three months new bone has filled the perilymphatic space. In some cases at five months this bone has become incorporated with the bone surrounding

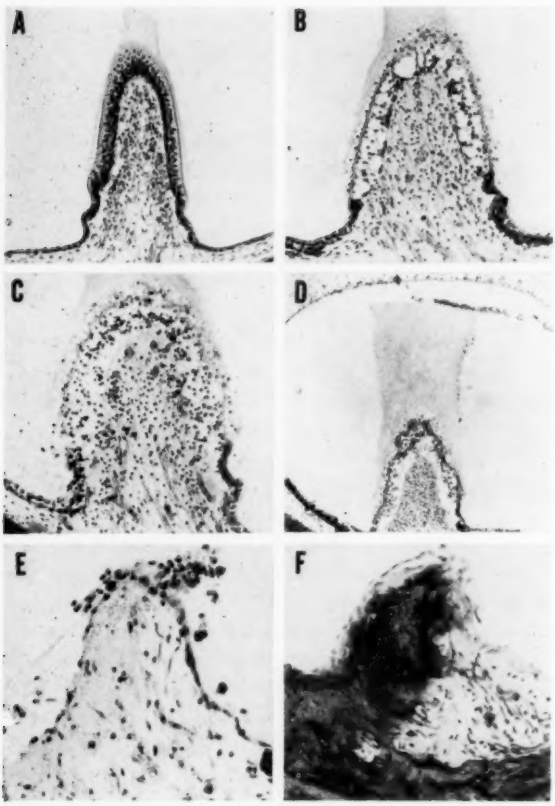

Fig. 7.—Progressive changes in the crista after arterial obstruction. A. Normal control. B. Edema and pyknosis of neuroepithelium at 16 hours. C. Advanced disintegration of neuroepithelium at 24 hours. D. Similar lesion showing cupular substance — remarkably free of shrinkage at six days. E. Complete loss of neuroepithelium at eight days. F. Ossification of crista at two months.

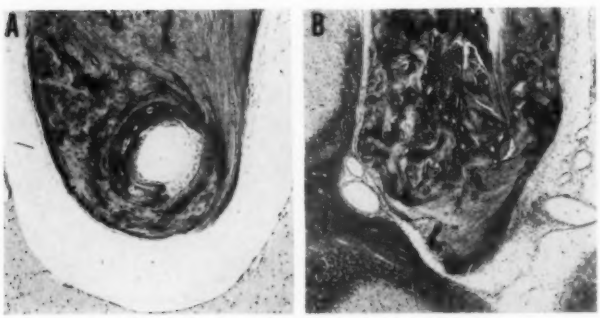

Fig. 8.—Section through superior semicircular canal at edge of parafloc-culus cavity showing stages in ossification after arterial obstruction. A. Ossification limited to perilymphatic space. B. Complete bony obliteration of canal and reorganization and fusion with adjacent cancellous bone.

the labyrinth to completely obliterate the labyrinth space (Fig. 8). Free blood cells were rarely seen in the region of the ampullae or canals.

Endolymphatic Duct and Sac (Fig. 9). The epithelium of the duct and sac including surface globules in the pars intermedia showed very little, if any, change following this lesion even after the rest of the labyrinth had become filled with fibrous tissue and bone. The entire contents of the sac often stained more deeply with eosin as compared to the normal control ear on the same section. Occasionally masses of dark granule containing cells were found in the sac. There was no change in the size of the sac except in a few instances when the distal portion of the sac appeared to be distended by pink staining precipitate several weeks after arterial obstruction.

Scarpa's Ganglion (Fig. 10). Reduction in Nissl substance of the ganglion cells was seen in one hour. Chromatolysis was evident four hours after this arterial lesion. A few hours later the cells became shrunken and began to disappear. However, total loss of cells did not occur even with widespread end organ degeneration. The cells in the ventral portion of the ganglion appear more regularly involved. Vestibular nerve fibers undergo changes simultaneously with those of the ganglion cells. Some of the vestibular nuclei are occasionally involved along with other areas of the brain stem due to interruption of their arterial supply.

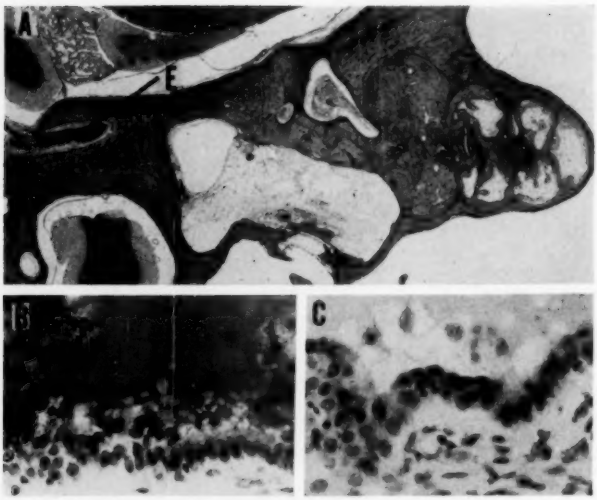

Fig. 9.—Section through entire labyrinth and endolymphatic sac showing advanced (5 months) reparative process after arterial obstruction. A. Cochlea filled with bone and vestibule filled with fibrous tissue. B. High power view of endolymphatic sac (E) showing dark staining contents. Compare with (C) same area of endolymphatic sac—control side.

Facial Nerve. The facial nerve fibers and geniculate ganglion were remarkably free from pathology after this arterial lesion even when the entire labyrinth was involved.

COMMENT

It is possible to interrupt the arterial supply to the vestibular labyrinth without surgical damage to the vestibular nerve or ganglion. This results in acute and sustained vestibular signs. The cold caloric test in the head position optimum for the horizontal canal gave results which corresponded to the histopathological findings.

An orderly progression of histopathologic changes could be established by examining specimens at various time intervals after acute arterial obstruction. Again the sensory cells are most vulnerable and show changes as readily as the hair cells in the organ of Corti. Various combinations of end organ involvement were noted. In one group all cochlear and vestibular end organs are involved; in the second, the

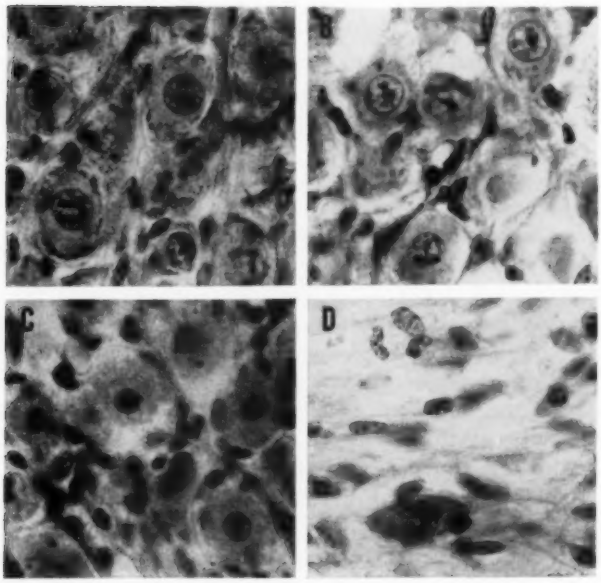

Fig. 10.—Sections through Scarpa's ganglion showing changes after arterial obstruction. A. Normal ganglion cells. B. Reduction in chromatin and Nissl substance and dissolution of cells at four hours. C. Pyknosis and reduction of Nissl substances at one day. D. Dissolution of ganglion cells at fourteen days.

cochlear, saccule and only the posterior ampulla are affected; in the third group, the utricle, horizontal and superior canal are affected along with part of the saccule but with no cochlear involvement. These variations in extent of damage following the surgical arterial interruption at the site of exposure can be explained by variations in the arterial branches actually occluded. The distribution of the lesions coincides with the arterial branches described by Nabeya[2] and by Smith[3] and indicate the possibility of arterial lesions in the human affecting only part of the vestibular labyrinth.

The early stages of edema and disintegration of the end organ resemble that seen in post-mortem degeneration but are more rapid because of the relation between temperature and rate of autolysis. The manner of separation of the sensory cell of the macula and crista from the supporting cell is unlike that of the organ of Corti. In the organ

of Corti the outer hair cells together with their supporting cells separate from the basement membrane. Only the inner hair cell is seen to separate from its supporting cell like those of the vestibular end organs. Widespread death of cells leaving only supporting ground substance usually followed arterial obstruction. However, some atypical changes such as retention of supporting cells with loss of the sensory cell, occasionally seen in our material, may possibly be explained on the basis of a temporary or partial obstruction. The reparative processes beginning at about one week and continuing to marked fibrosis and ossification are not unlike that seen after suppurative or chemical labyrinthitis. The absence of shrinkage of the cupular substance in arterial obstruction resembles that of the tectorial membrane. Similar changes are described in post-mortem degeneration and in chemical labyrinthitis. The cause for this is not clear. As in the cochlea the membranous vestibular labyrinth did not show signs of collapse or distension. Occasionally as with Reissner's membrane the utricular wall was denuded of cells; however, the saccular wall did not show this advanced change. The disposition of this cellular debris did not indicate endolymph movement. Similar to the findings in the cochlea, changes in the cells of Scarpa's ganglion and nerve fibers occurred as rapidly as those of the end organs and indicate a primary effect from interference with the blood supply to this area and not a secondary effect due to sensory cell degeneration. Moreover there was no evidence of direct surgical trauma to these cells. Unlike Rasmussen's bundle in the cochlea, the efferent facial nerve and geniculate ganglion are remarkably resistant to this arterial lesion. This may be due to the integrity of the facial nerve nuclei in the brain stem or preservation of the vascular supply to the geniculate ganglion.

Arterial obstruction produces more rapid and severe damage to the vestibular end organs than venous obstruction. The absence of hemorrhage after arterial obstruction is in striking contrast to the severe hemorrhage in the perilymphatic meshwork about the vestibular membranous labyrinth after venous obstruction. However, the subsequent fibrosis and ossification appears to be more severe and extensive after arterial obstruction. This difference is still evident at six months.

The pigment cells respond to this lesion in a manner similar to that of venous obstruction although pseudopodial forms are rarely seen. Unlike those found in venous obstruction, the cells of the planum semilunatum are often severely damaged by arterial obstruction. The cells of the utricular wall and ampulla adjacent to the pigment cells

are also severely damaged. After venous obstruction these cells are rarely affected and contain an abnormally large number of granules.

The preservation of the supporting cells of the vestibular end organs in some animals may be the result of slowed blood flow rather than sudden complete obstruction and resembles more the pathology seen after venous obstruction. The experimental procedure did not interfere with the venous return from the labyrinth. The integrity of the endolymphatic sac, even when the rest of the labyrinth was severely involved, was noteworthy. It indicates the independence of the blood supply of the sac from that of the rest of the labyrinth. Again as in cases of venous obstructions, the contents of the sac appeared to stain more deeply with eosin than the control side. In a few instances the distal end of the sac was distended. No concentrations of cellular debris were found in the sac. The epithelium of the sac wall and the concentration of globules were within normal range. The significance of these findings from the standpoint of explaining the function of the sac is not clear.

SUMMARY

Acute obstruction of the arteries to the vestibular labyrinth produces severe functional and pathological change.

Early irreversible damage resembles post-mortem degeneration. Later reparative processes ending in obliteration of the labyrinth by fibrous tissue and bone resembles that following labyrinthitis.

The degree and extent of damage was much greater than that following venous obstruction.

Lesions limited to only a part of the vestibular labyrinth are due possibly to the manner of branching the vestibular arteries.

The endolymphatic sac is not directly affected by this arterial lesion.

An orderly sequence of histological change was established and discounting post-mortem changes should serve as a guide in evaluation of human temporal bone sections with regard to arterial lesions of the vestibular labyrinth.

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II

COMPLICATIONS OF MASTOIDITIS IN THE ANTIBIOTIC ERA

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To the experienced otologist a paper on this subject may appear unnecessary. Having a pre-antibiotic memory, by which he steered himself through the early antibiotic years, he can sense the danger signs in his patient's course and "feel" the need for surgical intervention. To the newcomer in otology, the field is more vague. As Nilsen[1] pointed out, physicians in general have been "lulled into a relative dullness" by the decreased incidence of complications and the vagueness of their signs under the clouding of antibiotic therapy. The new otologist must learn to find the complications "hiding under a smog through which he must discern the diagnosis and effect a cure." For the sake of our students and house staff it appeared useful to review our experience of the past five years in the light of current literature and to crystallize for our department a present day philosophy of mastoid therapy. From such a study is drawn the material for this paper and to the needs of the student group this endeavor is devoted.

Recent literature gives ample evidence of the continuing occurrence of the complications of otitis media and mastoiditis, as illustrated by the reports of Shuster and Gordon,[2] Judge and Davis,[3] Tamari and Henna,[4] Maxwell,[5] Alexander et al.,[6] McLay,[7] Bastrup-Madsen and Norby,[8] and Morse.[24] In 1951 Davis[9] warned that mastoiditis following one or more previous attacks of suppurative otitis media, scarlet fever, measles, or so-called influenza requires careful observation for the onset of an intracranial complication. Courville's[10] recent study on the incidence of fatal complications is encouraging in that the deaths from this cause in a general autopsy series run about one-tenth as many as those of twenty years ago. Meningitis was the most common of these fatal complications. In his series thrombosis of the dural sinuses had been "practically eliminated"; there was an occasional brain abscess "adjacent" to the primary site, and only one "distant" abscess. Extradural and subdural abscesses were extremely rare and he observed

that these were usually secondary to abscesses in the brain. Of course, only a part of the reduction of these cases in an autopsy series would be due to an actual decrease of incidence. Much of the improved statistics may reflect successful therapy instead.

The most important step in the prevention of the complications is to be taken in the handling of the initial otitis media. The neglect of early myringotomy and the early abandonment of antibiotic treatment result from the hollow security offered by prompt symptomatic response to small doses of antibiotics. It is known to be frequent practice, at least in the rural area in which this writer lives, for acute otitis media to be treated by a single injection of penicillin with the matter of follow-up observation being left to the discretion of the patient. The sudden appearance of acute mastoiditis with or without complications one or two weeks after such "one-shot" management is doubtless familiar to many otologists, as illustrated in cases reported by Ersner, Alexander and Myers in 1952.[11] Davison[12] reported in 1955 that over a ten year period he had not had to do a simple mastoidectomy on any patient coming to him for the initial treatment of acute otitis media. He regularly used large doses of penicillin or other antibiotic, continuing it until the appearance and mobility of the drum were normal.

The second step in prevention is the proper detection and handling of uncomplicated mastoiditis. Cases showing the classical indications for surgery still occur, and reports such as that of Begley[13] in 1955 continue to demonstrate that the results from unhesitating operative intervention in these cases is rewarding. The masked or lingering mastoiditis, on the other hand, offers high challenge as to diagnosis and surgical decision. In such a case bone necrosis progresses despite apparent resolution of otitis media.[22] If overlooked, it may manifest itself by the sudden appearance of complications. This problem was not unknown in the pre-antibiotic era,[14] but unsuccessful antibiotic therapy has enhanced its incidence. Danger signs emphasized by Morrison[15] are malaise, low grade fever, anemia and leukocytosis. Richardson[16] also emphasizes the importance of night pain in and around the apparently healed ear. Spearman[21] clearly describes this syndrome.

In 1953 Nabo and Hajts[17] analyzed a series of penicillin-treated acute otitis media cases. Those in which the initial resolution was merely apparent remanifested themselves in three ways. One group simply had a recurrence of the ear symptoms in two to five weeks. A second group had sudden high temperature with mastoid tenderness.

A third had evidence of a break-through of suppuration to some area outside the mastoid with few or no symptoms. Repetition of penicillin in these cases was of no avail.

The management of these complications, once they are evident, is generally accepted as being the establishment of surgical drainage to loculated infection and the traditional surgical measures for controlling the mastoiditis itself. In only one group has there been exception taken to this principle; namely, otogenic meningitis. In 1944 Waring and Smith[18] took the attitude that unless there is obvious need for mastoidectomy it should be avoided, at least until the acute phase of the disease has passed. This change in policy is also reflected in the attitudes of Flippin,[6] who in 1948 advocated that all patients with pneumococcus meningitis, without pneumonia, should undergo mastoidectomy, but in 1953 advocated operating only on those patients in whom the primary site could be unequivocably established and, even then, not in the acute phase of the disease. In 1954, McLay[7] stated: "Since the introduction of penicillin it has not been necessary to carry out any operation other than paracentesis on cases of meningitis occurring with acute otitis media unaccompanied by mastoiditis." This illustrates the trend in thought, but such a generalization appeals to me as dangerous. Of his eight pre-antibiotic cases complicating acute otitis media, all were operated upon and four died; whereas of his four analogous cases treated with antibiotics, without operation, if I interpret his statistics correctly, one lived and three died. Perhaps a more significant feature of his study was the value of using both surgery and antibiotics in meningitis complicating chronic otitis media. Of five pre-antibiotic cases, all were operated and four died. Of 15 analogous cases treated by surgery and antibiotics, 13 survived.

The sweeping optimism leading to such blanket abandonment of surgery in otogenic meningitis is understandable in the light of the reduction of the mortality in the pneumococcus group from about 95 per cent to between 30 per cent and 50 per cent upon the introduction of the antibiotics. However, this 30 per cent or more still stands and its further reduction remains a challenge. An encouraging evaluation of the problem appears in a study by Bastrup-Madsen and Norby[8] in 1955. They felt that, with proper selection of cases, a bolder return to the use of surgery may be the road to reducing this mortality. They analyzed a series of 30 cases of pneumococcus meningitis. Most of their cases proved to be otogenic and they noted that the temporal bone osteitis which serves as the pathway of infection need not necessarily produce the clinical manifestations usually accepted as indications for

mastoidectomy. On penicillin alone (12,000,000 units per 24 hours), the mortality rate met the usual 30 per cent. However, they had no mortality in their series of ten cases in which they adopted the policy of doing mastoidectomy when rapid response to antibiotics was not obtained, in the absence of an evident primary focus outside the ear. They felt further that when a primary focus was not found outside the ear, the meningitis should be regarded as otogenic even though no ear abnormality is observed.

These authors took issue with the objection of Waring and Smith[18] to surgery during the acute phase of the disease. All seven of their operated cases were in very poor condition, and all became responsive and obviously improved very shortly after operation.

REPORT OF CASES AND COMMENTS

An analysis was made of 90 cases of mastoiditis admitted to two hospitals between September 1, 1951 and August 31, 1956. This series does not include the many cases of chronic otitis media with mastoiditis being treated as out-patients. The chronic cases included in this series are those for whom hospitalization was required to facilitate either intensive antibiotic treatment for acute exacerbations, minor operative procedures such as removal of aural polyps or major surgical intervention. Likewise, the series omits many cases of mild mastoiditis occurring in association with acute otitis media and treated as out-patients.

Of the 90 hospitalized patients, 43 had acute or subacute mastoiditis, and of these, 20 underwent mastoidectomy. The remaining 47 were classified as chronic and 28 of this group underwent mastoid surgery. Three of this latter group were admitted for revision of mastoid operations done many years before the beginning date of the present series. One of the chronic cases had operations on both ears.

In the 90 cases there were 21 instances of known complications, 11 among those classified as acute or subacute and 10 among the chronic. Not included in these complications is a case of brain abscess which had been present in the initial hospitalization of a patient now re-admitted for revision of the mastoidectmy. The complications found in this series were:

Meningitis . . . 9 cases

Laterial sinus thrombosis . . . 3 cases

Lateral sinus thrombosis with brain abscess . . . 1 case

Epidural abscess . . . 4 cases

Cerebritis . . . 1 case

Facial paralysis . . . 1 case

Subperiosteal abscess . . . 2 cases

They will be presented in four main groups: 1) meningitis, 2) epidural and lateral sinus infections, 3) cerebritis and brain abscess, and 4) other complications.

MENINGITIS

Of the nine instances of meningitis, four occurred with acute mastoiditis and five with acute exacerbation of chronic mastoiditis. No attempt has been made to review the general incidence of meningitis in these hospitals. This is important and warrants a separate study, because of the position taken by Bastrop-Madsen and Norby that any case of pneumococcus meningitis without a definite primary site outside the ears should be regarded as otogenic. The present study is limited, however, to those cases of meningitis carrying an established clinical diagnosis of mastoiditis.

The first two cases present the picture of meningitis occurring in the presence of known suppurative otitis media in small children, treated with antibiotics alone. In both, the meningitis responded but the mastoiditis did not and required further treatment, one finally having to have mastoidectomy soon afterward.

CASE 1. M. T., a six month old female infant, was admitted on December 15, 1953; having developed an upper respiratory infection three weeks before this date, with vomiting, hyperextension of the neck, and twitching of the hands. These symptoms had improved on terramycin. A few days prior to admission the vomiting had recurred along with somnolence and hyperextension of the neck. For two days she had again been on penicillin and terramycin.

On admission the temperature was 39 C, pulse 130, respiration 30. The infant reacted only to pain. It was in an opisthotonic posture. There was external strabismus on the left and a nystagmus to the right. The Babinski reflexes were positive bilaterally.

Laboratory findings included a hemoglobin of 9 grams and an erythrocyte count of 2,220,000, but the leukocyte count and differential were normal. The cerebrospinal fluid leukocyte count was 356 with 48 per cent polymorphonuclear neutrophiles. The total protein was 94 mg per cent and sugar 25 mg per cent. A smear revealed rare gram negative bacilli. The culture was positive for hemophilus influenzae.

Course in hospital: Streptomycin, gantrisin, and aureomycin were administered until December 28. Two days after admission a subdural tap was done obtaining 50 cc of purulent fluid from the right side, none from the left. Three weeks after admission, one week after stopping the antibiotics, she was noted to have discharge from the right ear. Culture revealed hemolytic Staphylococcus aureus which was sensitive to Chloromycetin,® which was then started. On February 17 a craniotomy was done by the neurosurgical service with removal of a subdural membrane diagnosed by the pathologist as the wall of a subdural abscess. The antibiotics were discontinued on February 26. The child was apathetic but was eating well and was afebrile. On March 9 she was again noted to have discharge from the right ear and an otologist was asked to see the case for the first time. There was found to be a large perforation in the right drum with heavy granulations in the middle ear cavity along with scanty purulent discharge. It was felt that this was a chronic otitis media of long standing, doubtless anteceding the present illness. Hemolytic Staphylococcus aureus was again cultured. It is not clear to this examiner why further treatment to the ear and mastoid were not done. The child was discharged on March 24 to be followed at the clinic but failed to reappear. Two and a half years later, at the time of the study, the family doctor was contacted. He stated that he did not know of further ear trouble in this case but he had noted that the child was seriously retarded mentally.

Case 2. T. T., a six months male infant, was admitted on December 7, 1954 with convulsions. Two and a half weeks prior to admission there had been discharge from the left ear for one day. The night before admission the infant developed a temperature of 103.6 F, followed by anorexia, vomiting, chills, convulsions, and a rigid position of the head.

Examination on admission revealed a temperature of 102.6 F, pulse 150. The left ear canal was full of white pus. The right ear drum was injected and full. Inspiratory crepitus and scattered rales were noted in both lung fields. There was increased resistance to passive movement of the right arm and both lower extremities.

Laboratory findings included a leukocyte count of 23,800, 83 per cent being polymorphonuclear neutrophiles. The cerebrospinal fluid cell count was 1,500 with 92 per cent polymorphonuclear neutrophiles. Smears revealed gram positive diplococci and the culture was positive for pneumococcus. X-ray of the chest was negative. X-rays of the mastoids revealed clouding and suspected cell breakdown on the left.

Course in hospital: Intensive penicillin and sulfonamide therapy was maintained for one week followed by achromycin for eight days. A left hemiplegia was noted on the third day. Bilateral subdural taps were negative. The infant gradually improved and was discharged on December 26 in good condition except for spastic paralysis of the left arm.

Two days after discharge from the hospital the infant developed a temperature of 103.5 F and was re-admitted. The respiratory rate was 70. There were fine and coarse rales throughout both lung fields and there was heavy mucopurulent discharge from the left ear. Culture from this ear revealed hemolytic Staphlococcus aureus and hemophilus influenzae. X-ray examination of the left mastoid revealed more clouding than on the previous admission. Heavy penicillin therapy was begun and on December 31 this was changed to achromycin. The first otological consultation was obtained on January 4. At that time there was deep reddening and fullness of the left drum with sagging of the posterior drum margin forward. Myringotomy obtained yellow mucopus under pressure. On January 7, a simple mastoidectomy was done. The cellular system had broken down forming large cavities, with edematous mucosa and some mucopurulent fluid. The dural and sinus plates were intact and were not opened. The child immediately improved. Three days later the drum was

flat, intact, and of good color. There was moderate drainage from the mastoid incision. On January 23 the child was discharged in good condition.

Whether in either of these cases early mastoidectomy would have altered the rate of recovery or the ultimate status of the patient is not answerable. Certainly in Case 2 the disease as a whole, including both the mastoiditis and the meningitis, was not finally controlled until surgery was done. In both, one might argue that the meningitis was unrelated to the otitis, but in the presence of so destructive a disease one would be reluctant to withhold any promising means of therapy where there is possible, if not probable, relevance. In the following adult case there is again the problem of a resolved meningitis but a persisting focus with the ever-present danger of recurrence:

Case 3. E. F. H., a 59 year old white male, was admitted on March 11, 1956 on transfer from another hospital. He gave a history of having had recurrent discharge from both ears with moderate deafness for 37 years. Three weeks prior to admission here he had developed severe headache, loud tinnitus in the right ear, vertigo, anorexia, and vomiting. He had a feeling "as if the ear broke," but there was no external drainage. On admission to the first hospital a lumbar puncture was done which showed an initial pressure of 270 mm with leukocyte count of 5,330, 88 per cent polymorphonuclear neutrophiles. He was treated with penicillin, streptomycin, sulfadiazine and, later, Mysteclin.®

On admission to this hospital the temperature was 37.5 C, pulse 80, respirations 22, blood pressure 120/80. The patient was alert and cooperative, but the gait was unsteady. There was minimum nystagmus on right and left lateral gaze. The hearing was impaired, more so on the right. There was increased resistance to passive flexion on the neck but no definite rigidity.

Laboratory findings included a blood leukocyte count of 19,550 with 64 per cent polymorphonuclear leukocytes. The cerebrospinal fluid had an initial pressure of 170 mm and a leukocyte count of 88, showing distinct improvement over the findings in the first hospital three weeks earlier. Culture from the right ear showed hemolytic Staphylococcus albus.

Course in hospital: The patient remained afebrile in the hospital and was given no specific therapy. He had no complaints except for dull headaches in the right temporal region which gradually subsided. An otological consultant saw the patient on March 13, 1956 and observed that the left drum was dull and retracted and that the right was discharging a heavy yellow pus. There was a suspected epitympanic perforation on the right. It was felt that the patient should have temporal bone surgery, but he has not returned here for this procedure, and it is not known whether he has had it done elsewhere.*

In the following four cases prompt surgery was rewarded by prompt uneventful recovery:

* After this report had been submitted for publication, this patient returned to the hospital with recurrence of meningitis and suppurative labyrinthitis. An endaural radical mastoidectomy and labyrinthectomy was done by Dr. J. W. Heisse, with subsequent recovery.

CASE 4. B. B., a 27 year old housewife, was admitted on February 4, 1952 with a chief complaint of headache. She had had ear attacks of brief duration off and on throughout her life, but each of these had completely subsided after one or two days of discharge. Three days prior to admission she had had an earache on the left side followed in a few hours by discharge from this ear and the onset of generalized headache. A few hours later she began to have nausea and next day the headache was more severe, her temperature was 102 F, and she was started on penicillin.

On admission the temperature was 100 F, pulse 60, blood pressure 122/80, respirations 24. The patient was crying because of severe headache. The neck was resistant to flexion. The Kernig's sign was positive bilaterally. The left ear drum was red and lacking in luster. The right ear appeared essentially negative. The physical examination was otherwise not abnormal.

Laboratory findings included a blood leukocyte count of 24,700 with 91 per cent polymorphonuclear neutrophiles. The cerebrospinal fluid leukocyte count was 2,370 with 91 per cent polymorphonuclear neutrophiles. A smear of the fluid revealed gram positive diplococci identified by culture as pneumococcus. Mastoid X-rays revealed bilateral sclerosis with a little more cloudiness on the left than the right.

Course in Hospital: An immediate simple mastoidectomy was done. She was found to have a necrotic area in the dural plate with granulations spread over the dura. There was softening of the periantral cells and much granulation tissue was found in the antral and attic areas. Postoperatively there was immediate general improvement and the cerebrospinal fluid leukocyte count one day post-operatively was only 503. The patient was kept on penicillin and sulfadiazine for two weeks and was discharged on February 21 in good condition.

CASE 5. F. K., a 42 year old white male, was admitted on January 31, 1955, complaining of pain behind the right ear and dizziness. He had bilateral ear abscesses as an infant and throughout his life had had discharge off and on from both sides. Three years prior to admission there had been one brief bout of pain in the right ear. Twelve days prior to admission the pain began behind the right ear followed three days later by the onset of frontal headache, vomiting, tinnitus, and an unsteady gait.

Examination on admission revealed a temperature of 97.6 F, pulse 98, respirations 20, blood pressure 144/82. There was a bilateral purulent discharge from the ears. The optic fundi revealed some blurring of the nasal borders of both discs. There was diplopia on gaze to the left, to the right, and upward. There was no nystagmus. The right corneal response was reduced. There was a marked reduction of hearing, especially on the left.

Laboratory findings included a leukocyte count of 15,500 with 65 per cent neutrophiles. The cerebrospinal fluid had an initial pressure of 280 mm with a leukocyte count of 1,530, with 160 of these being polymorphonuclear neutrophiles.

Course in hospital: There being clinical evidence of Gradenigo's syndrome on the right, with meningitis, a right radical mastoidectomy was done on February 8, 1955. A large attic cholesteatoma was found along with a fistula in the horizontal semicircular canal. The lateral sinus and dural plates were opened but no abnormalities were observed on these structures. Postoperatively the patient was maintained on chloramphenicol and sulfadiazine. He had a mild transitory right facial weakness which started one day postoperatively. By February 15 the cerebrospinal fluid leukocyte count had fallen to 272. He was discharged on March 3, 1955 in good condition.

CASE 6. R. L., a 15 year old white male, was admitted on April 22, 1955, complaining of pain in the right ear of four weeks' duration. He had had recurring

bouts of right ear pain since infancy and had had practically continuous discharge from this ear for three years. There had been pain with increased discharge for four weeks, right sided headache for two weeks, fever, anorexia, and vertigo for two days.

On examination the temperature was 100 F, pulse 100, respirations 22, blood pressure 116/74. The patient was quiet and oriented. There was a pulsating purulent discharge from the right ear. The drum was red and swollen, and there was a small low posterior perforation. The left drum was scarred, intact, and dry. The throat was hyperemic. The remainder of the examination was essentially negative.

Laboratory findings included a leukocyte count of 10,000 with 83 per cent neutrophiles. The cerebrospinal fluid revealed a total protein of 34 mg per 100 cc and a cell count of 29, mostly polymorphonuclear. The smear contained gram positive diplococci some of which were intracellular. A culture from the ear revealed a pure pneumococcus. X-rays of the mastoids revealed a blurring and lack of cellular detail throughout the right mastoid process.

Course in hospital: Patient was started on 1,000,000 units of penicillin every two hours along with chloramphenicol 1 gm every six hours. On the day after admission the temperature had reached 102.2 F. There was now bifrontal headache with nuchal resistance. On the second day a simple mastoidectomy was done. Thin exudate was found in many cells and frank pus was observed in the antrum area. There were some granulations on the dura in the middle cranial fossa and some heavy granulations around the lateral dural sinus. However, the sinus itself did not seem to be thrombosed. On the day after operation the patient was afebrile and the following day he was free of headache. By May 5 the drum was entirely healthy in appearance, and the patient was discharged on May 8.

CASE 7. E. R., a 53 year old white male, was admitted on August 16, 1956 with a chief complaint of headache of three weeks' duration. It had been increasing steadily and 48 hours before admission he developed malaise, weakness, nausea, and vomiting. On the day prior to entering the hospital he had chills and fever. The patient had been having a painless persistent discharge from the left ear for many years.

Examination revealed a temperature of 101 F, pulse 100, respirations 20, blood pressure 130/80. The patient was anxious, tense, sweating, and complaining of chilliness. There was a serosanguinous exudate in the left ear canal. There was neck pain on flexion. He also had a slight hyperalgesia of the left side of the face.

Laboratory findings included a leukocyte count of 17,500 with 66 per cent neutrophiles. The cerebrospinal fluid on admission revealed a total protein of 154 mg per cent, an erythrocyte count of 1200 (not crenated), and a leukocyte count of 700 with 80 per cent polymorphonuclear. An ear culture obtained Proteus mirabilis. X-ray examination of the mastoids revealed bilateral sclerotic changes.

Course in hospital. Otologic consultation revealed that the right ear drum was filled with a large polyp surrounded by a thin yellow fluid. The clinical improvement on streptomycin and chloramphenicol was poor. On August 22 an endaural radical mastoidectomy was done. There was erosion of the dural plate and a cholesteatoma filled the epitympanic area, its matrix being against the dura. Because of the complete relief of symptoms the patient was discharged on September 3 still having 210 cells (99 per cent lymphocytes) in the cerebrospinal fluid.

Two cases are now presented in which the antibiotics evidently controlled both the meningitis and the mastoiditis. In one of these

the meningitis was of a sterile "irritative" type and the mastoiditis early.

CASE 8. M. H., a five year old girl, was admitted on February 21, 1955 with the complaint of fever and lethargy. Three weeks prior to admission she had become irritable, feverish, and coughing and for three days her temperature had been up to 104 F. She had vomited several times and complained of earache. She was given achromycin and penicillin for the three days prior to admission.

Examination revealed a temperature of 40 C, pulse 129, respirations 30. The child was acutely ill, irritable, and lethargic. The neck and back were moderately stiff. She had positive Kernig's and Brudzinski's signs bilaterally. Both ear drums were red and swollen. There was no mastoid tenderness. The pharynx was moderately injected. The rest of the examination was essentially negative.

Laboratory findings included a leukocyte count of 35,000 with 40 per cent neutrophiles, 55 per cent lymphocytes and 6 per cent monocytes. The cerebrospinal fluid leukocyte count was 210 with 90 per cent lymphocytes. No growth was obtained on culture. Mastoid x-rays revealed a diffuse clouding bilaterally.

Course in hospital: The child was put on intensive penicillin therapy amounting to about 3,000,000 units per day. The temperature was normal within 36 hours. She became gradually more alert and less irritable. The ear drums became less red and less full. She was sent home on March 9, 1955 on sulfadiazine.

The child was readmitted on May 17 because of having had a recurrence of earache, fever, and irritability eleven days previously. This had been treated with penicillin and achromycin, but the ear drum still appeared inflamed. At this time the first otological consultation on this case was requested. It was believed that the x-rays now appeared improved and that the left otitis media was subsiding. She was again treated with penicillin, 1,200,000 units per day, and was discharged in three days, the ears having apparently cleared.

CASE 9. H. L., a 37 year old female was well until five weeks prior to admission when she noted the rapid onset of pain in the left ear followed a few days later by a bloody purulent discharge. She also had a slight fever. She was given penicillin and erythromycin for a few days and was admitted to a hospital elsewhere. A lumbar puncture was done revealing 300 cells, at least 75 per cent of which were polymorphonuclear. A culture of hemophilus influenzae was obtained. The patient was given a twelve day course of intensive streptomycin and chloramphenicol therapy and was transferred to this hospital.

Physical examination on admission revealed a temperature of 37 C, pulse 84, respirations 16. She walked with a wide unsteady gait. There was a large central scotoma on the left side. There was blurring of the disc margins, and bilateral weakness of the external rectus with diplopia in all fields of horizontal gaze. The facial musculature was more active on the right than on the left. The hearing was poor bilaterally, especially on the right.

Laboratory findings included a leukocyte count of 8,750 with 84 per cent neutrophiles. X-rays of the mastoids revealed slight clouding on the left as compared with the right.

Course in hospital: An otological consultant found that the right drum was deeply retracted and a little red. There was no perforation. There was a severe perceptive hearing loss. The left drum was intact, retracted, and on this side there was a moderate perceptive hearing loss. It was suspected that the source of the meningitis had been the left ear, the deafness on the right being a result of the

meningitis. A simple mastoidectomy was done on the left. The mastoid cells themselves were essentially clean. A small exposure in the dural plate and another in the lateral sinus plate failed to show any granulations on either of these structures. She was maintained on chloramphenicol for about ten days and discharged improved.

The surgery in Case 9 proved unnecessary. It was done primarily because of spinal fluid manometrics suggesting lateral sinus obstruction, but this was not found. In this case antibiotics had cleared the mastoiditis. The lack of chronicity, the nearly concomitant onset of the otitis and meningitis, the healthy appearance of the ear drums after antibiotics and, to some degree, the fact that the infecting organism was hemophilius influenzae, were features weighing against surgical intervention.

These few cases seem to bear out the value of early mastoid surgery in otogenic meningitis where the ear infection is well developed prior to the onset of meningeal signs. We are also reminded that where antibiotics may control the meningitis they may fail to eradicate the mastoid disease, allowing the danger of recurrent complications to continue. Our finding of epidural and perisinus granulations in the operated cases is a serious warning to the physician who does not consider prompt surgery in otogenic meningitis.

EPIDURAL AND LATERAL SINUS INFECTIONS

This group consists of seven cases which present various stages of the development of infection in the lateral sinus area, ranging from bony erosion with heavy granulations upon the sinus and dura, up through purulent perisinus and epidural abscesses, to three cases of fully developed septic thrombi within the vessel. In four of these there was epidural involvement of the middle cranial fossa as well. In one there was also a temporal lobe abscess, this case being the only one in the entire study with a fatal outcome. In the following two cases the perisinus infection was not suspected but was found in operations being done on the strength of pre-antibiotic classical indications in acute mastoiditis:

CASE 10. T. O., a seven year old boy, was admitted on February 13, 1952. He had had a right earache during an attack of measles one month prior to admission. This had subsided and then ten days before admission it had recurred with discharge. Then the left ear also developed a discharge eight days later. On the day before admission the child was noted to have a bulging over the right mastoid process. One injection of penicillin was given.

Examination on admission revealed a temperature of 100.4 F, pulse 100, respirations 20, blood pressure 108/70. There was protrusion of the right ear with swelling

and tenderness over the mastoids. There was purulent discharge from both ears. Both drums were red and bulging.

Laboratory findings included a leukocyte count of 19,000 with 72 per cent neutrophiles. X-ray revealed probable erosive changes in the superior cells of the right mastoid process.

Course in hospital: Immediately after admission a myringotomy and a simple mastoidectomy on the right side were done. All mastoid cells were found to be filled with granulations. The upper third of the lateral sinus plate was destroyed and the sinus was covered with a plastic exudate. Culture from this area revealed hemolytic Staphylococcus and beta hemolytic Streptococcus. The child was kept on penicillin and gantrisin postoperatively with the latter being replaced on the 12th day by terramycin. The postoperative improvement was rapid. Within two days both drums appeared normal and the retroauricular discharge ceased in about a week.

Case 11. C. W., a five year old girl, was admitted to the hospital on May 13, 1955 with earache of three days' duration on the left side, increasing despite penicillin therapy. For two days there had been nausea and vomiting.

Examination on admission revealed a temperature of 102 F, pulse 120, and blood pressure 110/65. The right ear was essentially negative. The left revealed a pink, thick, dull, bulging ear drum with a tiny posterior perforation through which there came a pulsating purulent discharge. There was redness, edema, and tenderness over the mastoid. Examination otherwise was essentially negative.

Laboratory findings included a leukocyte count of 19,300 with 75 per cent neutrophiles. A culture from the left ear revealed Proteus and a few hemolytic Staphylococcus areus. X-ray examination revealed diffuse clouding of the left mastoid.

Course in hospital: Myringotomy on admission obtained abundant serosanguineous discharge. She was treated with gantrisin and streptomycin, but there being no improvement in two days a simple mastoidectomy was done. Yellow pus was found beneath the periosteum, although no definite cortical perforation was seen. Most of the cells contained pus. The lateral sinus and dural plates were both eroded and there were heavy granulations over both the sinus and the dura. The sinus was not firm and was believed not to be thrombosed. Postoperative improvement was rapid. Within six days the drum appeared to be of normal color and position.

It is noteworthy that if these cases had been treated by antibiotics alone, and had responded well, we would have congratulated ourselves on a conservative cure but would have unwittingly left a masked intracranial spread. This is especially disturbing when we know that in our total series there were at least eight acute mastoiditis cases in small children apparently "successfully" treated without surgery. (Subacute cases are not included.)

In the following young people the surgery was not based on full classical indications, but on moderate or even tenuous suspicions with the rewarding discovery that the procedure thwarted serious intracranial spread:

CASE 12. A. B., a 25 year old housewife, was admitted on October 13, 1954, with the complaint of tenderness behind the right ear. She had had chronic right otorrhea since childhood without any medical attention. About three and a half weeks prior to admission she had been hospitalized on the medical service for an upper respiratory infection with a flare-up of the pain and discharge in the ear and severe right temporal headache. She had been treated with antibiotics with relief of the headache and was discharged in ten days on ear drops. The aching in the ear continued. She was seen in the otology clinic for the first time two weeks later where the previous event was interpreted as possibly representing an intracranial spread of infection from the mastoid, followed by masking with the antibiotics. On the strength of this opinion she was readmitted.

Examination revealed a pale chronically ill young woman with a temperature of 98 F, pulse 80, respirations 18, blood pressure 110/78. The left ear drum was retracted and intact. The right canal contained a brown, crusted discharge. There was evidence of an attic perforation with widespread destruction. Redness and tenderness were noted over the mastoid process. The remainder of the examination was essentially negative.

Laboratory findings included a leukocyte count of 9,650 with 72 per cent neutrophiles. The cerebrospinal fluid findings were essentially negative. A culture from the right ear revealed Pseudomonas aeruginosa.

Course in hospital: A lumbar punction on the day of admission revealed an initial pressure of 230 mm with manometrics suggestive of a right lateral sinus thrombosis. X-ray of the mastoids revealed sclerosis around the right antrum and in the inferior portion of the mastoid process there was an oval area of decreased density. The patient was started on penicillin and streptomycin. Two days after admission a radical mastoidectomy was done with the finding of a large thrombus in the lateral sinus. This was followed about halfway to the torcular sinuum before fresh blood was encountered. There were many granulations around the sinus but no definite pus either in or around the vessel. The dural plate was also eroded and there was heavy granulation tissue upon the dura but no free pus. Much pus was encountered in the antrum and tympanic and epitympanic areas. An internal jugular vein ligation was done. Postoperatively the course was excellent except for a few days of slight dizziness and headache. She was discharged on November 12 in good condition.

CASE 13. D. C., a 12 year old boy, was admitted on March 31, 1955, with the chief complaint of earache of two weeks' duration. He had had an intermittent discharge from the left ear for about five years. Two weeks prior to admission he began to complain of left earache and the drainage increased in quantity. He complained of soreness in the left side of the neck. He was admitted to another hospital where hot wet compresses were applied and ear drops used but was discharged in two days. He continued to complain of pain. There was an increasing swelling of the area around the ear and he had some tendency to vertigo.

Examination revealed considerable edema above and behind the left ear. The canal contained mucopurulent discharge. The drum was thick and scarred. There was a large posterior inferior perforation filled with granulations. There was no mastoid tenderness. The remainder of the physical examination was essentially noncontributory. There was no leukocytosis.

Course in hospital: Despite the streptomycin and erythromycin therapy the edema over the mastoid increased and on April 2 a radical mastoidectomy was done with a finding of a large cholesteatoma which had eroded both the sinus and dural plates. There was epidural granulation over the temporal lobe area as well as in the posterior cranial fossa over the sinus. The postoperative course was excellent. The child was discharged on April 16 with a clean mastoid cavity.

CASE 14. A. S., a 13 year old girl, was admitted on January 8, 1956, complaining of left earache and headache. Five days before admission she had the onset of left earache during an upper respiratory infection followed two days later by discharge from this ear. On the day prior to admission she began to have severe bifrontal headaches. Has had about two attacks of left otorrhea per winter, usually following colds.

On examination the temperature was found to be 98.2 F, the pulse 120, respirations 23, blood pressure 120/70. The left ear drum was red and full. There was profuse drainage seeming to come from a low anterior perforation. Although there was no edema over the mastoid, there was rather marked tenderness which extended for about 4 cm superoposteriorly to the antrum area. Otherwise the physical examination was essentially negative.

Laboratory findings included a leukocyte count of 18,000 with 82 per cent neutrophiles. Culture from the left ear revealed a beta hemolytic Streptococcus. X-ray of the mastoids revealed diffused clouding on the left side with probable cell erosion.

Course in hospital: The patient was started on penicillin and streptomycin. Three days after admission there was still mastoid tenderness although the middle ear findings were improved. Lumbar puncture revealed an initial pressure of 160 with manometrics suggestive of a partial occlusion of the left lateral sinus. The next day a simple mastoidectomy was done. There was a cortical erosion with granulations extending through to the subperiosteal area not from the mastoid itself but from the perisinus area. There was widespread necrosis of the sinus plate. Heavy granulations lay upon the sinus and well posteriorly there was encountered a flow of pus from between the bone and the dural sinus. The vessel itself did not seem grossly thrombosed. Three days postoperatively the patient was fully alert and comfortable. The drainage persisted for nine days. She was discharged on the twelfth postoperative day in good condition.

In the surgical decisions on all three of these patients much weight was given to the sudden appearance of unusual acute features upon a pre-existent history of infection. Beyond this the signs were meager. In Case 12 there was only a mild redness and tenderness, but the bout of severe headache with fever which the patient had recently undergone was interpreted as an intracranial spread. Although Case 13 had postauricular swelling, there were few supporting findings, there being no fever, leukocytosis, or tenderness. The vertigo, though only mild, was a warning. Case 14 presented bifrontal headache, which was considered suspicious. Here again, it could well have been disastrous to have tided these cases along with antibiotics.

The last two of this group were definitely quite ill and the full-fledged septic thrombosis was anticipated preoperatively:

CASE 15. R. D., a 48 year old white male, was admitted on January 20, 1952, with a complaint of fever and chills. He had had an intermittent discharge from the right ear for 40 years. Three weeks prior to admission he had had mild right ear pain with an increase in the discharge. Two days prior to admission he felt chilly, followed by four episodes of definite chills with a temperature spiking up to 104 F.

Examination on admission revealed a temperature of 101 F, pulse 100, respirations 22. The right drum was full and thick. There was a small posterior perforation with pulsating purulent discharge. There was edema over the mastoid process and tenderness over the sinodural angle area with extension of the tenderness directly posteriorly, to the occiput. The neck was not resistant to movement. There were a few transient nystagmoid movements especially on right lateral gaze. The remainder of the examination was essentially negative.

Laboratory findings included a leukocyte count of 17,700 with 85 per cent neutrophiles. Culture from the right ear revealed Proteus morgani. The same organism was found at operations in the intracranial infection. X-ray revealed a widespread breakdown of the right mastoid with an oval shaped area of decreased density suggesting an opening in the lateral sinus plate.

Course in hospital: The patient was started on Gantrisin® and penicillin and a myringotomy was done on admission. The following day a simple mastoidectomy was done. On opening the cortex a mass of foul grey pus with gas bubbles was encountered. This material came chiefly from a huge abscess occupying the lumen of the lateral sinus. There was also a large periantral cavity containing cheesy white material with much yellow pus. The lateral sinus was exposed for about 6 centimeters of its length before a free blood flow was obtained. A ligation of the right internal jugular vein was then done. After operation there was apparently a steady clinical improvement for several weeks. Then the patient began to show evidences of mental change. The spinal fluid pressure remained elevated. Electro-encephalography on February 26 revealed possible frontal lobe changes. A revision operation was done with conversion to a radical mastoid cavity. The dura of the middle cranial fossa was noted to bulge into the cavity and to be covered with granulations, but no direct tract could be found into the temporal lobe. Ventriculography the following day suggested brain abscess. Two abscesses were believed located and drained in the right frontal lobe by needling by the neurosurgical department, but the patient became steadily worse and died on February 28. Autopsy revealed a huge abscess cavity occupying the right temporal lobe.

CASE 16. M. T., a 20 year old white male, was admitted on May 21, 1954, with the complaint of right sided headache of about three weeks' duration. Eight years previously the patient had had a brief episode of right ear pain and discharge followed by intermittent otorrhea ever since. Three years prior to the present event he had had severe pain and heavy discharge with a diffuse throbbing headache. This had persisted for six weeks before he sought medical attention, at which time a small operative procedure was done in the ear canal and he was kept on penicillin for many weeks. The present episode began with an upper respiratory infection three weeks prior to admission accompanied by a severe right sided headache, persisting until admission. There was no ear dicharge.

On examination the temperature was found to be 37.4 C, pulse 96, respirations 36, blood pressure 135/78. There was tenderness to palpation in the right mastoid and occipital areas. The fundus of the right ear canal was filled with a polyp around which there was a yellow, sour, purulent exudate. The remainder of the physical examination was essentially non-contributory.

Laboratory findings included a leukocyte count of 18,150 with 87 per cent neutrophiles. X-ray examination revealed widespread sclerosis of the right mastoid with an area of rarefaction in the periantral area and a larger area of decreased density more posteriorly. No cell structure could be delineated. Culture of the right ear obtained Proteus vulgaris.

Course in hospital: The patient was started on erythromycin. On the day after admission the temperature spiked to 38.6 C and he was noted to have a left homonom-

ous hemianopsia along with some left facial weakness. A lumbar puncture obtained a fluid under 300 mm of initial pressure with a cell count of 9 leukocytes. On the following day a simple mastoidectomy was done. The initial penetration of the cortex led to a large abscess cavity. This proved to be in the perisinus area just posterior to the usual location of the sinus plate. The periantral area contained pus and granulations. The dura of the middle cranial fossa appeared to be negative. There were heavy granulations and fibrin formations around the lateral sinus extending to three centimeters posteriorly to the mastoid area itself. A thrombus was found and was

removed. A pocket of green-grey pus in the epidural space just superior to the sinus was opened. Heavy discharge persisted for thirty days from the postauricular wound and then gradually subsided. The patient slowly returned to full alertness and well being. On June 18 electroencephalogram had showed marked abnormalities in most of the right hemisphere. On this day a re-exploration of the mastoid cavity was done with conversion to a radical cavity. Foul smelling purulent discharge was found in the epitympanic and middle ear areas. The patient was discharged after 57 days in the hospital with a clean dry cavity. He was seen two months later and was still in excellent condition.

It may be of real significance in Case 16 that three years prior to the present event the boy had gone through a bout of ear pain and heavy discharge with diffuse throbbing headache treated over many weeks with penicillin. In the light of the other cases there seems little doubt that he should have had his emergency surgery then. Better yet, of course, would have been surgical control of the chronic disease at any earlier stage. It is known that Case 15 had been repeatedly advised over a 15 year period to have a radical mastoidectomy.

CEREBRITIS AND BRAIN ABSCESS

The only instance of a fully developed brain abscess in the series is in Case 15 presented above. The clinical evidence of the abscess was vague and only came into evidence many weeks after the initial operation for lateral sinus thrombosis. It seemed probable from the autopsy specimen that its beginnings had anteceded the first operation. In any event, there is no doubt that the abscess grew right under our supervision and in the presence of huge doses of antibiotics. It appears likely in retrospect that transmastoid drainage in the second operation would have been a better approach than needling by burr-hole, even though a tract was sought and not found in the operation. Localization by electroencephalography and by neurological examination had been unsuccessful. Brain infection was suspected but it was assumed to be on the basis of retrograde thrombophlebitis rather than by extension to the temporal lobe. In a second instance, summarized below, clinical localization was again tenuous, but the outcome more fortunate.

CASE 17. C. L., a 50 year old white male, was admitted on April 9, 1956, in a state of coma. The patient was known to have had discharge from both ears for about twenty years, more consistently from the left than from the right. Two days prior to admission he began to complain of pain behind the left ear and began vomiting and became lethargic.

Examination on admission revealed a patient in coma, rousing only to painful stimulation. There was nuchal rigidity. The left upper and lower extremities were moved perhaps more freely than those on the right. All deep reflexes were exaggerated, slightly more so in the right. The left plantar reflex was flexor and the right equivocal. Exudate was noted in both external ear canals. The right drum showed deep red discoloration in Schrapnel's membrane with granulations visible in this area. The left drum was deeply retracted and scarred. There was a probable superoposterior marginal perforation. The exudate was scanty and almost dry. The remainder of the examination was essentially negative.

Laboratory findings included a leukocyte count of 13,900 with 76 per cent neutrophiles. A lumbar puncture obtained an initial pressure of 210 mm, a protein of 87 mg per cent, sugar 74 mg per cent, and cell count of 206, half being erythrocytes and half leukocytes with 87 per cent polymorphonuclear. X-rays of the mastoid processes revealed poor pneumatization bilaterally with periantral sclerosis. On the right there was clouding and loss of cellular detail. A defect was noted in the upper margin of the right petrous pyramid suggesting breakdown.

Course in hospital: With the patient in coma and with both ears obviously infected it was difficult to determine a rational surgical course. An electroencephalogram showed some questionable evidence of a focus in the left tempora area. The history of left ear and eye pain in the few hours preceding loss of consciousness was helpful. A few hours after admission a left radical mastoidectomy was done. As the exploration approached the base of the petrous pyramid between the horizontal and superior semicircular canals there suddenly gushed forth a flow of thin yellow opaque fluid under pressure. It was not possible to demonstrate definitely a fistulous tract. It was believed to have come from the base of the petrous just above the horizontal semicircular canal. Because of persisting coma a left-sided angiogram was done three days later revealing some elevation of the left middle cerebral artery. Consequently, on April 13 an exploratory burr-hole was put in over the left temporal area where the neurosurgeons encountered some cerebritis but no definite abscess. The ventricular fluid was found to be sterile and to have a cell count of 426 of which only 35 were leukocytes, the rest being erythrocytes. There was then a slow but progressive improvement. By April 18 he had become quite alert but had a total VI nerve paralysis on the left. There was pain on the left side of the face and hypalgesia in the second and third divisions of the left trigeminal nerve. He was retained on achromycin throughout his hospital stay. He was discharged on May 5. He was followed for four months thereafter, showing steady improvement.

This case is probably best interpreted as a suppurative petrositis with Gradenigo's syndrome and a fulminating cerebritis, doubtless on its way to the formation of a temporal lobe abscess.

OTHER COMPLICATIONS

The two instances of subperiostial abscess and the one of facial paralysis were all in young children with acute, rapidly developing mastoiditis, all presenting classical indications for surgery and all

responding promptly to simple mastoidectomy. Their only relationship to the problem of antibiotics was the failure of each child to respond to antibiotic therapy in the few days prior to hospitalization. They only serve to remind us that the acute disease, in its old classical form, is not entirely eliminated.

SUMMARY AND CONCLUSIONS

It is clear from this study of our own cases and the related literature that dangerous complications of mastoiditis are continuing to occur. Often they are the penalty of incautious reliance on antibiotics alone in earlier stages of the disease. Once they occur they may be insidious and difficult to diagnose under the cloak of antibiotic therapy. It appears wise to go ahead with mastoidectomy if any of the classical signs of acute mastoiditis are present; such as, postauricular swelling, mastoid tenderness, sagging of the posterior canal wall or unquestioned x-ray evidence of bony breakdown. This statement is made in spite of the fact that our own series includes 22 cases of acute and subacute mastoiditis treated with antibiotics alone. Our warning here is based on the finding of unanticipated intracranial extension in operated cases. Furthermore, surgery appears to become indicated when an acute mastoiditis has subsided on antibiotic therapy but there is a lingering on of unexplained malaise, low-grade fever, anemia, leukocytosis, pain in and around the ear, headache or vertigo.

In meningitis associated with otitis media the possibility of mastoid surgery should be kept constantly in mind and becomes indicated when the patient's progress is not prompt and satisfactory. A non-surgical management with intensive antibiotic therapy appears justified when the meningitis and the otitis are of simultaneous onset. If, however, an ear infection seems to respond well to antibiotics but is followed in a week or more by meningitis, a masked mastoiditis should be suspected whether or not there is recurrent otorrhea, and surgery becomes indicated. In the light of this review it appears that at least three groups of meningitis cases call for consideration of mastoid surgery:

1. Those with delayed onset in the course of acute otitis media.

2. Those with early onset in acute otitis media but resistant to antibiotic therapy.

3. Those coming in the course of chronic otitis media.

A salient feature in the prevention of dangerous complications is, of course, the proper control of chronic otitis media. It is not in the

scope of this paper to deal with the methods of such control and their indications. It is, however, relevant to this study to point up dangerous situations, demanding prompt surgery, as over against ordinary exacerbations in the chronic problems, in which delay is permissible, conservative treatment worthy of trial, and surgery scheduled when convenient. In the presence of an acute exacerbation of chronic otitis media, certain features must be taken as warnings of serious complications. Pain in the ear, in a patient whose usual exacerbations are painless, is a danger sign. Pain above and behind the ear is even more significant. Mastoid tenderness, and especially tenderness superoposteriorly to the mastoid, should be given serious weight in a chronic case. Headache, even if only frontal, is important in these cases. General malaise, listlessness, anorexia, slight temperature elevation, and slight leukocytosis should be looked on with suspicion. Even mild degrees of vertigo or ataxia, changes of mentation or clouding of consciousness should be respected. Above all, the observer should follow up even subtle suspicions with adequate neurological examination, lumbar punctures, electroenophalography and other studies, and should be ready to operate on thin evidence. The spiking fevers and leukocytosis of classical description may fail to appear.

An interesting observation in these cases is the finding of Proteus in three, and Pseudomonas in one, of the most serious cases as the chief causative agent. Although these organisms are often found in chronic ear infections, their ability to produce dangerous complications is worthy of note.

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III

INTRALARYNGEAL INTRATRACHEAL THYROID

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Thyroid tissue inside the larynx and trachea is rare, to the extent that none of several texts, devoted entirely to the thyroid gland, even mention its existence in such a location. Nevertheless, such a case encountered by the author aroused enough interest to stimulate careful scrutiny of the literature in an attempt to determine its pathogenesis and frequency of occurrence.

EMBRYOLOGY

The thyroid gland arises from the ventral floor of the pharynx as an invagination of entoderm, at the level of the first pharyngeal pouch. This diverticulum of thyroid tissue lies at the bifurcation of the aortic trunk, and becomes a solid mass which may be bilobed as early as the fourth week of fetal existence. At the same time, the thyroglossal duct is forming from the pharyngeal floor, connecting with the base of the tongue at what will become the foramen cecum, this being, ordinarily, the only remains after the thyroglossal duct atrophies during the fifth week. The so-called descent is actually a result of the forward growth of the pharynx, with the gland settling in a transverse position on the trachea with a lobe on each side in its final position by the seventh week. The solid bilobed thyroid mass begins to acquire colloid after the eighth week of fetal life, and the conversion into follicles is finished by the sixteenth week. After this, new follicles form by budding.

THEORIES OF PATHOGENESIS

Bruns[1] believed that a part of the embryonic thyroid gland becomes completely separated from the main mass, and is displaced into the interior of the larynx and trachea. There has been no scientific data to substantiate this theory.

Fig. 1.—Lateral roentgenogram of neck showing encroachment upon the subglottic airway.

Paltauf[2] stated his belief that the thyroid gland grows into the larynx and trachea during extra-uterine life, especially during puberty. He cited his case of a 29 year old female in which he could prove that the intra- and extratracheal thyroid had a direct connection between the cricoid and first tracheal ring.

Wegelin[3] agreed that there is always a bridge of thyroid tissue between the external thyroid and the intralaryngeal thyroid, which perforates the cricotracheal membrane, but he insisted that this occurs not only in extrauterine life but also during fetal development. He examined several white rats, in which goiters had been produced by a special diet. Intralaryngotracheal thyroid tumors were found in several of these rats, and a bridge between the internal and external thyroid tissue was definitely illustrated in five. In rats without external goiter, he found no intralaryngeal thyroid tissue. From this investigation, he concluded that hypertrophy and hyperplasia of the external thyroid gland favors invasion of the larynx and trachea.

Falk[4] also maintained that there is invariably a bridge between the external thyroid and the intralaryngotracheal mass, but from his investigations, he concluded that thyroid tissue does not penetrate the larynx and trachea postnatally, but rather that the internal mass always has its beginning during intrauterine life. To substantiate this belief, he made sections of twenty-one larynges of prematures and newborns, and found thyroid tissue beneath the laryngeal mucosa in nine. In one of these, there was an intralaryngeal goiter development. In all of these cases, the intralaryngotracheal thyroid was found on the posterior wall. Falk felt this was due to the lesser resistance to penetration in this area, where there is only loose connective tissue and mucous glands, as compared to the tough and adherent submucosa anteriorly. He pointed out that the ingrowing of thyroid tissue is, also, especially favorable during fetal life when the thyroid gland is not encapsulated. The intralaryngotracheal mass cannot be considered true aberrant thyroid tissue if a direct connection with the external thyroid is established. The cases cited above of Paltauf, Wegelin, and Falk would, therefore, be considered false aberrant goiters.

Beeson[5] reported a case of intralarynegal thyroid tumor as "being attached to the right vocal cord and its vicinity." The microscopic pathology "showed the tumor to consist entirely of thyroid tissue, with no evidence of accompanying malignant growth." This case would sustain the theory of Bruns.

To explain the occurrence of true aberrant intralaryngotracheal thyroid tissue, one would have to consider that, quite early in the development of the embryo, the oral cavity bends farther ventrally than is ordinarily believed, with movement of the thyroid into the immediate vicinity of the fourth branchial arch, thus allowing actual penetration of thyroid tissue into the larynx.

At the present time, it is generally agreed among most authorities[6] that lateral ectopic thyroid rests are actually a metastasis from the gland proper, and represent metastatic malignant goiter. In most of those cases in which it has been possible to carefully study the main thyroid gland, or to observe the ultimate outcome, a malignant process has been proven in the adjacent lobe of the thyroid.

Therefore, a nonmalignant thyroid tumor inside the larynx or trachea must have its pathogenesis by: 1) direct extension from the thyroid gland through the cricotracheal membrane (false aberrant thyroid) or 2) deposit of thyroid tissue early in the development of the embryo during descent of the thyroid gland (true aberrant thyroid).

Fig. 2.—Repeat roentgenogram taken seven months after Figure 1 revealing even greater narrowing of airway. Barium outlines hypopharynx.

INCIDENCE

Intralaryngotracheal goiter is, admittedly, a condition of considerable rarity. Yet, the investigations of Wegelin[3] and Falk[4] would indicate that the presence of thyroid tissue in this location is common, and its potential development into a goiter not a remote possibility. Most of the reported cases came to clinical attention because of mechanical obstruction to the airway. It is safe to assume that many escape the notice of the clinician or pathologist because they have not grown to the extent of causing symptoms.

Since the development of the intralaryngeal goiter is usually associated with hyperplasia and hypertrophy of the external thyroid gland (Wegelin[3]), it would be expected to occur more frequently in the geographical regions where goiter is more common.

DIAGNOSIS

As in most cases of partial upper respiratory obstruction in which the voice is not affected, the first sign is a wheeze. This, almost invari-

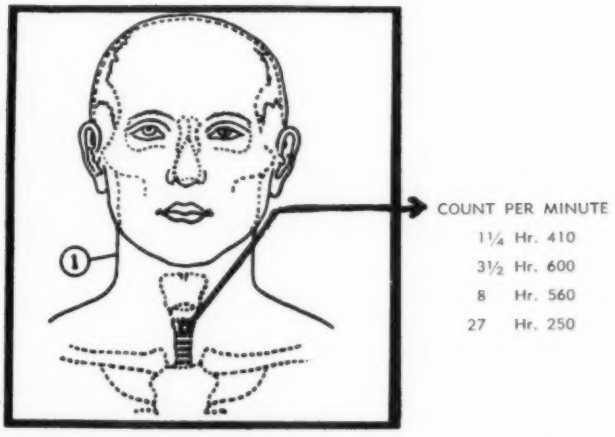

Fig. 3.—Geiger counter chart showing localization of thyroid tissue after tracer dose of radioactive iodine.

ably, occasions an erroneous diagnosis of asthma. During the period of ineffective asthmatic treatment, the respiratory obstruction increases, until the embarrassment to the airway stimulates a direct examination of the larynx and trachea. Or, finally the stage of dyspnea, independent of exertion, is reached where a tracheotomy becomes a necessity.

A lateral roentgen study of the neck will show a narrowing of the airway in the involved area. Direct laryngoscopy and tracheoscopy will reveal the presence of a subglottic tumor. As has been suggested previously, this is usually a rounded, smooth mass arising from the posterior wall of the larynx and trachea. Suspicion that the tumor might consist of thyroid tissue is strengthened if an external goiter is also present. Biopsy of the mass will usually reveal considerable vascularity, and the histologic examination will show islands of thyroid tissue and mucous glands.

Chevalier Jackson,[7] in his mention of intralaryngeal thyroid, states that: "In the larynx and trachea, an island or a tumor of thyroid tissue is almost certain to take on malignant qualities sooner or later." He describes, however, two cases of thyroid tumor in the larynx with wheezing and dyspnea which had previously been treated for asthma, which had no evidence of malignant change in the microscopic sections.

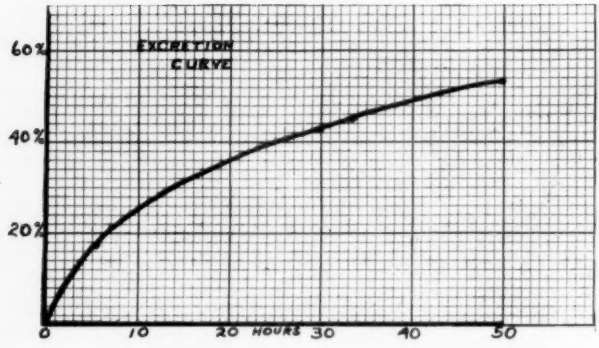

Fig. 4.—Excretion curve of radioactive iodine study suggesting malignancy.

Since there is frequent disagreement amongst reliable pathologists as to the presence, or degree of malignancy present, in the histopathology of any given case, study with a tracer dose of radioactive iodine may help to establish the existence of a malignant process in thyroid disease.

TREATMENT

Surgical removal of the intralaryngotracheal thyroid tumor is the procedure of choice. Even if it is not causing an embarrassment to the airway, the possibility of a benign tumor becoming malignant is indication enough for its eradication. This can perhaps be accomplished satisfactorily through direct laryngoscopy and tracheoscopy, with the aid of electrocoagulation to control the vacularity of the lesion. However, a better approach for complete removal of the larger tumor is by thyrotomy.

REPORT OF A CASE

The past history of Miss G. K. revealed that an enlargement in the thyroid area had first been noted at age six. A subtotal thyroidectomy had been done in 1945, at age 16, because of symptoms of hyperthyroidism. Following this, she had been treated with Lugol's solution, but because of poor control of symptoms, a right thyroid lobectomy was done in April, 1947. Subsequently, a wheeze developed, and

since she also had manifestations of seasonal pollen allergies, she was treated for asthma. The wheeze, and beginning respiratory obstruction, did not improve with allergic treatment, and the patient was again hospitalized in May, 1948. A lateral roentgen study of the neck (Fig. 1) showed a marked subglottic constriction of the airway, with the encroachment coming from posteriorly. The roentgenologist stated that he could not determine whether the narrowed airway was due to an intralaryngotracheal lesion, or from a retrotracheal mass. A left hemithyroidectomy was done, but when the endotracheal tube was removed in the recovery room, marked inspiratory difficulty was encountered. I was called for my first examination of the patient, and direct laryngoscopy immediately revealed the posterior subglottic swelling. After the endotracheal tube had been reinserted, a tracheotomy was done with immediate relief of the dyspnea.

The endotracheal tube had apparently caused just enough irritative swelling, at the site of the tumor mass, to completely close off the narrowed airway. When this swelling subsided, it was possible to decannulate her, but the wheeze and mild dyspnea persisted. Because of the reluctance on the part of the patient and her parents, towards further surgical procedures, she was discharged from the hospital on May 18, 1948.

However, the slight dyspnea gradually progressed to severe upper respiratory obstruction, with marked retraction at the suprasternal notch, intercostal spaces, and epigastrium. She finally consented to hospital readmission on December 27, 1948. A tracheotomy was repeated, and direct laryngoscopy again revealed the smooth, rounded tumor arising from the posterior and right lateral walls of the subglottic and upper tracheal regions.

A lateral roentgenogram of the neck portrayed even greater encroachment on the airway as compared to the study done seven months previously (Fig. 2).

Since it was felt that the tumor mass was probably related to the external hyperplastic thyroid gland which had been present before her thyroid operations, a tracer study was done using 50 microcuries of radioactive iodine (I^{131}). This examination identified thyroid tissue in the location of the intralaryngeal and intratracheal mass (Fig. 3). The tracer pattern suggested a malignant process in the thyroid tissue, since the excretion curve was still rising thirty hours after the administration of the radioactive iodine (Fig. 4). The study also showed that the thyroid tissue had almost normal intrinsic iodine function

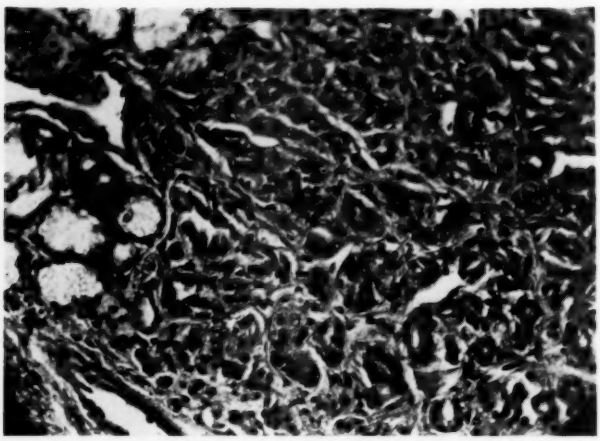

Fig. 5.—Histopathologic section showing thyroid tissue with invasive growth into area of mucous glands.

with a very satisfactory retention, making the patient a suitable candidate for isotope therapy if other forms of treatment were found unsatisfactory.

Following these examinations, a thyrotomy was done. With adequate exposure, the mass was found to arise from the posterior and right lateral wall of the subglottic area, beginning about one centimeter below the right vocal cord and extending down to the second tracheal ring. The tissue was very vascular and not encapsulated. It was dissected away from the submucosa of the larynx and trachea, but it appeared to invade between the tracheal rings at its lower level. It was therefore felt that exenteration of the mass in this area had perhaps been incomplete.

A frozen section report was returned of "intralaryngeal thyroid carcinoma."

To insure complete removal of the tumor, it appeared that it would be necessary to remove the larynx and a section of trachea. Since the patient was only nineteen years of age, and since the I^{131} tracer study suggested that the lesion might respond satisfactorily to isotope therapy, it was decided to remove the mass as completely as

Fig. 6.—Lateral roentgenogram of neck showing adequate airway one year after removal of intralaryngotracheal thyroid tumor.

possible, while leaving the larynx and trachea intact, thus avoiding a permanent tracheal stoma in so young a girl.

Hemostasis was accomplished, and the larynx was closed, leaving the tracheotomy tube in situ. Postoperative course was uneventful, and the tracheotomy tube was removed five days after operation with a completely adequate airway. She was discharged from the hospital on January 22, 1949, sixteen days after her thyrotomy operation.

Routine pathologic section was reported as substantiating the frozen section report of "intralaryngeal thyroid carcinoma" (Fig. 5).

She was rehospitalized on February 28, 1949, for direct laryngoscopy which revealed healing to be complete with no evidence of airway encroachment.

Considering the histopathologic report of thyroid carcinoma, and the possibility of incomplete removal of the tumor from between the

tracheal rings, it was decided to treat her with radioactive iodine in an attempt to destroy all remaining thyroid tissue. Consequently, the patient was given a total of 182 millicuries of radioactive iodine in small, divided doses between March, 1949 and March, 1950. Progress of the therapy was observed by repeated tracer studies in which, finally, no residual thyroid tissue could be detected. Lateral roentgen study of her neck one year after thyrotomy revealed an airway approaching normal (Fig. 6).

Regular clinical check-up examinations have not revealed any evidence of recurrence of the intralaryngotracheal mass, and repeat radioactive iodine tracer studies done at yearly intervals show no regeneration of thyroid tissue to the present time. Her myxedema has been satisfactorily controlled by three to four grains of thyroid extract daily. The patient is carrying on normal activities, with regular working hours, and has been apparently happily married for six years.

COMMENT

Histopathologic diagnosis of thyroid cancer is, at the present time, in a state of considerable disagreement. The very reputable pathologist who made the diagnosis of "intralaryngeal thyroid carcinoma" over seven years ago in the case just reported, in a recent review of the microscopic section, reversed his diagnosis, and now calls it "thyroid adenoma." Yet, a review of the slide by other competent pathologists, in preparation for this report, has resulted in opinions of the presence of malignancy while others have judged it benign.

In this state of confusion, it would seem advisable to treat these questionable cases, at least, as potential malignancies with as complete eradication of the lesion as is practicably possible.

Surgical treatment of the thyroid gland, in its normal location, does not come within the province of the otolaryngologist in this geographical location. Nevertheless, since other neck surgery is within his domain, he will occasionally encounter lateral masses of thyroid tissue. With the prevailing concept that these are all malignant metastases from the thyroid gland proper, it becomes important that all of these lesions be found (they may be multiple in a single patient), and the use of radioactive iodine tracer studies has proven a great aid to their discovery.

The intralaryngotracheal thyroid, described in this paper, does not necessarily fall into this category of a metastatic lesion, since its

location on the posterior wall would suggest that it may have been a direct extension through, from the thyroid gland proper, as suggested by the German investigators. A review of the microscopic sections from her previous thyroid operations failed to show evidence of malignancy.

CONCLUSIONS

1. Intralaryngotracheal goiter is a condition of considerable rarity, but should be kept in mind as a distinct possibility in diagnosis of tumors in this location, especially when a concomitant hyperplasia of the external thyroid gland exists.

2. A tracer study with radioactive iodine is an important adjunct to diagnosis, and will help to direct the proper course of treatment, especially in the face of the current controversial histopathologic diagnosis.

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IV

THE WHISPERED VOICE

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GRONINGEN

The whispered voice was frequently used in phonetics especially in the days of the Helmholtz-Herrmann controversy. If the vowels were produced by whispered voice, then the registration of the various frequencies was not impeded by the low strong basic tone. In the audiological department of the O. R. L. Clinic in Groningen (Prof. Dr. H. C. Huizing), it was possible, thanks to modern apparatus and the use of 1/3 octave filters, to make an accurate analysis of the vocals as produced by conversational and by whispered voice. There is always some difference between the vowels of different persons and also between vowels of the same person at different times. But it is a well-known fact that each vowel has a typical pattern. Figures 1 and 2 are chosen as good average examples of the Dutch aa and the Dutch oo, spoken with normal and whispered voice. There is some difference in the middle frequencies (which will be left undiscussed), but naturally the most striking feature is the lack of lower frequencies in the vocals by whispered voice.

There is not only a quantitative but also a qualitative difference between the conversational and the whispered voice. Although this has always been taken into account in phonetics, it has not hitherto sufficiently penetrated acoustic investigations. This must probably be explained by a certain routine, based on the fact that the whispered voice was used in determining auditory acuity long before the development of audiology. It is a very simple method of investigation, and it must be admitted that it does afford a gross impression of the degree of hearing impairment and also more or less of the nature of the hearing impairment (conductive or perceptive). This is possible by using words chiefly consisting of low components (zona gravis) and words in which high components predominate (zona acuta). The first to take into account the difference in high and low frequencies in words of whispered speech were the Dutch investigators, Zwaardemaker and Quix.[1] This is a good example of the closely knit connection between phonetics and audiology.

Communication at the Voice Conference in Chicago, May, 1957.

The results obtained, however, are quantitatively very unreliable. In this country this has been pointed out by Fowler[2] and by Glorig.[3] The intensity of whispered speech showed considerable variations with various investigators and in the same investigator at different times. It is regrettable that the authorities in many countries still attach such great importance to investigations using whispered speech. The fitness requirements for certain occupations are still often expressed in terms of the ability to hear the whispered voice at a certain distance. This gives rise to extremely unfair decisions because the various forms of hearing loss are associated with a relatively better or less good hearing of the whispered voice.

Whispered speech is used because investigations using the conversational voice are, as a rule, impracticable. With the development of electrical apparatus this has considerably improved since the introduction of speech audiometry with the aid of P. B. lists.

The audiologist, Huizing, and my former assistant, Kruisinga,[4] have made an investigation into speech audiometry with whispered voice in a manner similar to that which has become a routine method with the conversational voice in all audiological centers. The results were remarkable. The shape for the curves for the spoken voice and for the whispered voice is the same, but the curve of the whispered voice starts at a lower intensity. It passes the threshold of the intelligibility (50 per cent) at the 20 db level, whereas this threshold for the spoken voice is about 10 db higher. Contrary to what was hitherto assumed, at very low intensities whispered voice is much more understandable in normal hearing than spoken voice.

There is a very characteristic difference between patients with a conductive and patients with a perceptive deafness. In conductive deafness the articulation curves are merely shifted to the right; the two curves have still the same relationship as the curves in normal hearing. In perceptive deafness, however, the relative position of the curves is changed. Opposite to the curves of whispered voice in normal hearing and conductive deafness the curve for whispered voice in perceptive deafness runs at higher intensities. This phenomenon is called by Huizing *vocal inversion.*

The phenomenon of vocal inversion is characterized by the fact that in contrast to normal the spoken voice is understood at lower intensities than the whispered voice. The explanation is simple. In perceptive deafness the higher frequencies are impaired. At the same

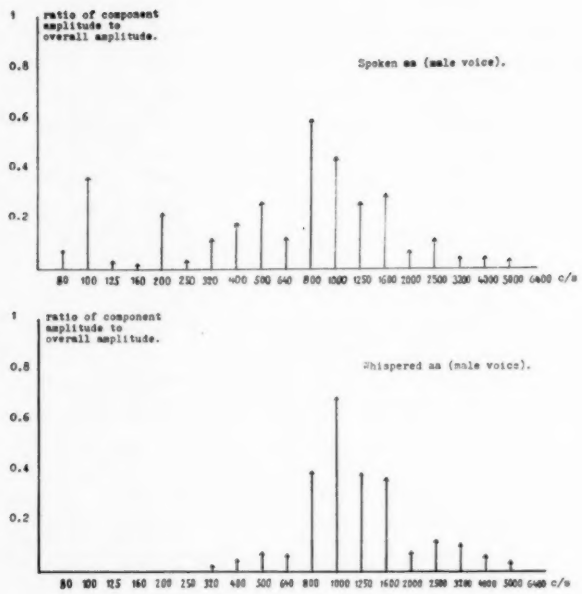

Fig. 1.—aa with normal and whispered voice. The different frequencies are, as in Figure 2, given as the ratio of the overall amplitude.

time there is the lack of low components in the whispered voice. Apparently the combination of these two factors can no longer be compensated for by the redundancy of clues, which normally also is present in whispered voice for discrimination of the different speech sounds.

It is clear that the whispered voice test may not be used as a reliable index for the social hearing capacity. But if it is applied on real principles it can give a valuable contribution for auditory and speech training in a certain percentage of hard of hearing children.

An interesting experiment was done by Huizing with a two channel band pass filter. When speech is processed through this circuit two groups of selected components in the low (140 to 280 c/s) and medium (1128 to 2256 c/s) range are boosted up. Starting at zero position of the attenuator and gradually increasing the amount of attenuation we find a remarkable difference in change of quality

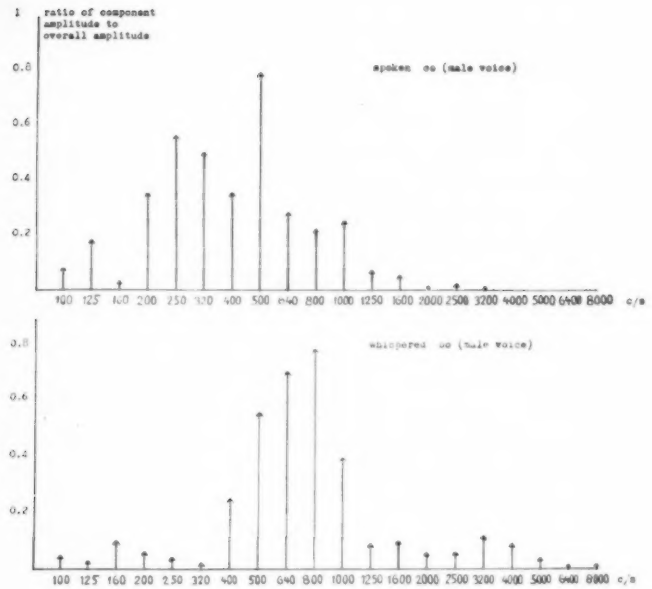

Fig. 2.—oo with normal and whispered voice.

of the spoken voice as compared to the whispered voice when speaking into the microphone. The intelligibility of the spoken voice is highly involved as soon as the attenuation of the medium range is introduced, although we find no considerable change in intensity. The essential higher components are blurred out by the strong low ones making the speech more and more distorted and hard to understand. On the contrary the whispered voice, though getting weaker and weaker, remains clear with a high grade of intelligibility.

The masking effect by the lower tones during the attenuation in conversational speech is the cause of this striking result. Under normal conditions in normal hearing there is no masking effect because the threshold of this lower octave is 30 to 40 db higher than that of the essential middle octave. The importance of this attribute of our ear is evident; it makes the discrimination of speech sounds possible.

This experiment has important consequence for the acquisition of speech and language by young children when they have to cope

with a hearing handicap characterized by a sloping audiogram at any grade of hearing loss. In such cases the child is more easily accessible by means of whispered voice if it is applied in the right way.

CONCLUSION

The use of the whispered voice in testing auditory acuity has often been criticized. Attention is drawn to experiments of Huizing and Kruisinga. They show once more that whispered voice cannot be used in official examinations. The strong low components are lacking in the whispered voice. Two typical examples are given of the various frequencies in vowels produced in conversational and in whispered speech.

With reference to the investigations made by Huizing and Kruisinga the consequences of this difference are pointed out. Patients with perception deafness are relatively less capable of hearing whispered speech. The lack of strong, low components makes the use of whispered speech a recommendable aid in the acquisition of speech and language in some children suffering from hardness of hearing.

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V

SURGICAL TREATMENT OF LABYRINTHINE DISHARMONY

JOHN J. BALLENGER, M.D.

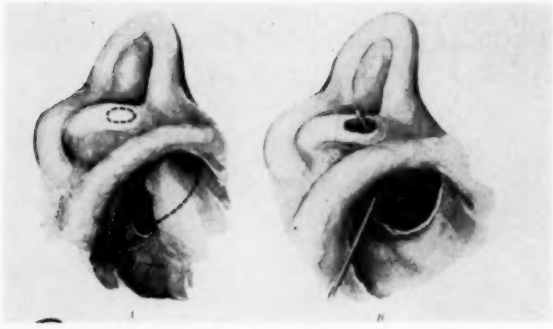
WINNETKA, ILL.

Suppurative labyrinthitis has become vastly less prevalent, fortunately, since the advent of the sulfanilamides and antibiotics. In discussing suppurative labyrinthitis I am confining myself to the labyrinth itself and its direct complications. This excludes, principally, petrositis and apicitis.

Labyrinthitis may be classified into serous, circumscribed and purulent varieties for purposes of this discussion. The first named probably is a clinical entity though this is a moot point. Presumably exotoxins gain entrance to the labyrinth and bring about disfunction by some means. The source of the toxins may be at some distance from the ear. In circumscribed labyrinthitis there usually is a fistula in the external semicircular canal. In neither of these is there suppuration usually. Their possible surgical treatment will be considered subsequently.

The incidence of suppurative or purulent labyrinthitis has been vastly reduced in recent years so that a discussion must draw heavily on the experiences of the past. In former years the presence of suppuration in the labyrinth or suggestive intracranial spread of a preexisting labyrinthitis indicated, and rightly so, immediate operation to drain the mastoid or the labyrinth. This was carried out without delay, regardless of the hour. Since the advent of the sulfonamides and particularly the antibiotics the surgeon is entitled to wait and administer these drugs in an attempt to localize or perhaps eradicate the suppurative process. It is not clear in my mind how long one may wait. Fortunately I have had too few cases. Were signs of meningeal irritation to persist despite adequate antibiotic therapy I would be tempted to intervene surgically.

From the Department of Otolaryngology, Northwestern University Medical School and Evanston Hospital, Evanston, Illinois. Read before the Alumni Meeting of the Brooklyn Eye and Ear Hospital April 27, 1957, Brooklyn, New York.



A *B*

Fig. 1.—Hinsberg's operation. *A*. The plate of bone, separating the oval and round windows, to be removed. *B*. A probe has been inserted through the newly created opening into the vestibule and out through the opening into the lateral canal. The facial ridge separates the two openings.

It is generally agreed I believe that destructive labyrinthotomy for suppuration is not indicated, nowadays, as long as any vestige of vestibular or cochlear activity remains. Any co-existing suppurative process, however, which would usually be the case, even though slight, in the mastoid and middle ear or petrous apex should be subjected to wide drainage by means of a mastoidectomy or apeceotomy.

Once this has been done and labyrinthotomy has been decided upon there are several operations to choose from. As accurate a diagnosis as possible of the primary site of the suppuration in the labyrinth should be made. Of the many operations which have been devised I shall describe only three because by their use all parts of the labyrinth may be reached and the source of the pus directly attacked.

The approach may be either post- or endaural, followed by a complete tympanomastoidectomy if not already done as a preparation for each of the techniques to be described. The resulting tympanic portion should have a wide flat appearance with as low a ridge of bone over the facial nerve as is consistent with safety. The superior posterior walls of the external auditory canal, the external attic wall and the anterior lip of the eustachian tube are beveled back until the oval and round window are brought into view.

In the Hinsberg operation,[1] the vestibule, the lateral semicircular canal, and the cochlea are opened without exposure of the dura. Hence, the labyrinthine spaces only are drained. The end result is illustrated in Figure 1. As you can see the lateral canal has been opened, the round and oval window joined, and the promontory removed so as to open the lower whorl of the cochlea.

Jansen has described two operations. In the first of these the lateral canal is opened and followed forward until the vestibule is centered. Through this opening the orifices of the other two canals are exposed as well as the basal turn of the cochlea.

Jansen's second operation[1] has been modified by Neumann and is known as the Jansen-Neumann operation. Following the procedure as described above, the dura over Trautmann's triangle is exposed and elevated from the posterior wall of the petrous pyramid. Elevation continues until the internal auditory meatus is reached. Then the posterior surface of the petrous pyramid is removed until posterior and lateral semicircular canals are cut across. A probe inserted into the lateral canal will pass into the vestibule. The Jansen-Neumann operation drains the labyrinth and the meningeal spaces. Whether such extensive procedures are required now that we have the aid of antibiotics is yet to be decided.

Richards' operation also was an extensive procedure. Both the cochlear and vestibular labyrinths were opened as indicated in Figure 2.

No recently accumulated statistics are available to suggest the prognosis following labyrinthotomy for suppurative labyrinthitis or which is the best technique. In the three recent cases with which I am familiar all survived following a much less extensive operation.

I wish now to pass to a discussion of the surgery of nonsuppurative labyrinthine disharmony. This is certainly a more vital and alive subject. Perhaps the simplest instance of this surgery is a paracetesis of the drum for an acute otitis media. The opening of an obstructed eustachian tube by catheterization perhaps should be included in this category or the incision and drainage of an abscess anywhere which leads to a resolution of the source of a toxic labyrinthitis.

Much more tangible is the surgical treatment of the vertigo caused by a fistula. The fistula usually lies between the middle ear and the lateral semicircular canal, usually is due to chronic infection

Fig. 2.—Richards' operation. The apex of the cochlea is identified and the apical whorl opened. The middle whorl and the modiolus are not disturbed unless drainage of the meninges by way of the internal auditory canal is thought advisable.

or a cholesteatoma, and a radical or simple mastoidectomy with certain reservations is the accepted treatment.

It must be remembered that the fistula does not actually enter the peri- or endolymphatic space of the lateral semicircular canal. The fistula extends from the middle ear as far as the endosteum and thus is not a true fistula. The presence of a positive fistula test indicates a functioning labyrinth and thus labyrinthotomy is not indicated. The surgical treatment of the fistula must avoid probing the fistula or otherwise running the risk of extending the fistula into the peri- or endolymphatic spaces. If a cholesteatoma is present it is not disturbed in the region of the fistula and I personally do not cover the fistula with a skin graft.

Tinnitus must be included in a discussion of labyrinthine disharmony. I do not propose to enter into the interesting discussions relative to its etiology. One of the surgical attempts to relieve tinnitus was performed in New York by Dr. Lempert.[2] In 10 out of 15 patients he succeeded in relieving severe tinnitus of over one year's duration by avulsing the tympanic plexus.

Leighton F. Johnson[3] believed he relieved some cases of tinnitus by the injection of procaine into the stellate ganglion. If, after a

few minutes, relief ensured, a dorsal sympathectomy was advised in which the sympathetic roots of T2, and T3, and T4 were interrupted.

The technique of injection of the stellate ganglion is as follows. After introducing a No. 22 spinal needle at a point situated approximately 4 cm from the midline and 4 cm above the clavicle it is directed backward, medially and slightly downward until it strikes the transverse process of the seventh cervical vertebrae. Usually injection of 7-9 cc of 1 per cent procaine in this region is sufficient to ablate the ganglion as suggested by a good Horner's syndrome.

Following Dr. Johnson's unfortunate death the work was continued by Strong who reported[32] the effect of section of T2, T3, and T4 on tinnitus. Of four cases two have been relieved for two or three years. I shall have occasion to refer to this later.

Lewis[34] found that in his hands cervical sympathectomy abolished or satisfactorily relieved five of 17 cases. The operation consisted either of two types: preganglionic section of the sympathetic roots of T2, T3, and T4 or stellate ganglionectomy.

Cawthorne and Hewlett[20] have failed to note lasting benefits from injections of the stellate ganglion. Hoogland[23] on the contrary advises stellate ganglion block before destructive labyrinthotomy. In 37 cases not responding to medical therapy 60 per cent were relieved by one or more injections.

Another type of labyrinthine disharmony to which surgical attention has been directed is the "syndrome of non-syphilitic interstitial keratitis and vestibulo-auditory symptoms" described by David Cogan.[4] He reported four cases, the first of whom underwent a cervical ganglionectomy without relief of the auditory or ocular symptoms in the first three months. Mogan and Baumgartner[5,6] reported two cases of what seems the same disease process several years before. Both of their cases underwent cervical sympathectomy with improvement in the vertigo. Henry L. Williams[7] has added a seventh case and I have had one such case at the Evanston Hospital in Evanston, Illinois. In all of them the presence of an interstitial keratitis associated with severe vertigo is the essential picture. As the vertigo gradually lessens deafness of varying degrees supervenes and remains permanently. Removal of the sympathetic supply to the inner ear by surgical means may have a part to play here but I rather doubt it. Williams suggests that these cases may be the result of intoxication, perhaps with streptomycin.

When considering surgery of nonsuppurative labyrinthine disharmony the most interesting entity is, of course, intractable vertigo sometimes associated with Ménière's disease. Cawthorne[7] says 25 per cent of the patients with this affliction that he sees require some form of surgery. Williams[7] reports that at the Mayo Clinic this figure is 20 per cent and Lindsay's[8] 10 per cent at the University of Chicago. My own figures are more in keeping with Dr. Lindsay's. In most instances the surgical approach is applicable only to the 85 per cent of cases which are unilateral. Only very rarely can a patient with bilateral destructive labyrinthotomy walk unaided, drive a car, etc. Such a procedure is rarely justified especially if, as is reported, the second ear is much easier to control medically after unilateral labyrinthotomy.

I wish to discuss the following surgical approaches to relief from intractable vertigo in this order. First the surgery of the VIII cranial nerve itself which was inaugurated by Dandy in 1928, but improved by Dr. McNally's countryman, K. G. McKenzie, when he performed the first partial section of the VIII nerve in 1931. I wish then to pass to a consideration of the surgery of the sympathetic nervous system for this condition and for completeness' sake to mention alcohol injection of the labyrinth and surgery of the chorda tympani. Lastly I wish to speak about the partial and complete destruction of the labyrinth.

Barber and Ireland[9] in 1952 reviewed McKenzie's cases operated on between 1931 and 1951. Green and Douglas[10] performed a similar service for Dandy. These reports plus those of Castellano[11] and of Lathrop,[12] who gave the experience at the Lahey Clinic have been reviewed. It seems evident that partial section of the VIII nerve will relieve the vertigo in 70 to 90 per cent of cases and that total section about the same.

The tinnitus is relieved at best in 16 to 27 per cent whether by total or partial section. As will be pointed out later destruction of the labyrinth does equally as well as nerve section in relieving the vertigo and much better in relieving the tinnitus than nerve section. When considering hearing the balance tilts in favor of nerve section as compared to destructive labyrinthotomy. McKenzie's cases retained what Barber and Ireland call "useful hearing" for ten years in two out of three cases of partial section. Labyrinthotomy almost invariably destroys the hearing. It must be remembered, too, that nerve section is a formidable procedure with a mortality rate of approximately one per cent. I think nerve section should be reserved for

those cases in which preservation of the hearing is important and in which tinnitus is not a great factor.

Surgery of the sympathetic nervous system is based on somewhat tenuous grounds. It is presumed that vasoconstriction of the labyrinthine vessels occurs following activity of the sympathetic nerves and that the resulting ischemia is followed by vertigo and perhaps tinnitus. There is some evidence that the vascular supply of the labyrinth can be altered by tampering with the autonomic nerves. I wish to review this very briefly.

Frazier[13] used a dye injection technique. In an anesthetized cat dye was injected intravenously. Five seconds after beginning the injection the cervical sympathetic nerve or ganglion was stimulated. Sixty seconds after the onset of the injection the animal was bled to death. Staining was then found to be absent in the homolateral temporal bone and explained by the vasoconstriction caused by the stimulated sympathetic nerves. Stimulation of the stellate ganglion, after destroying the tympanic plexus, resulted in no vasoconstriction. In the search for the presence of dye the temporal bone was sectioned and the labyrinth studied microscopically in only part of the cases so I believe the accuracy of this report is open to question.

Wever[14] and his associates in 1940 noted in cats a reduction in cochlear microphonic potentials when the O_2 tension was reduced to 4 per cent on lower. Seymour and Tappin[15] have observed two things after stimulation of the cervical sympathetics. First there was a reduction in the cochlear microphonic potentials and second an invagination of Reissner's membrane. This is illustrated in Figure 3. This they thought was due to a reduction in the production of endolymph. Later due to osmotic changes they considered that the endolymph might be increased and the picture of endolymphatic hydrops created. It is difficult to say whether spasm, perhaps of the stria vacularis, resulted from stimulation of the cervical sympathetic trunk had caused these changes though Seymour and Tappin believe so.

Anatomically the autonomic nerve supply to the labyrinth is still a moot point. Many people believe, however, that to isolate the labyrinth from the sympathetic nerve supply is very difficult if not impossible. The sympathetic nerves reaching the circle of Willis via the internal carotid artery, the basalar artery and the vertebral artery probably must be cut. Garnett-Passe[16] believes cutting the sympathetic roots of T2, T3 and T4 is sufficient. He reports having observed in humans undergoing fenestration dilation of blood vessels of the

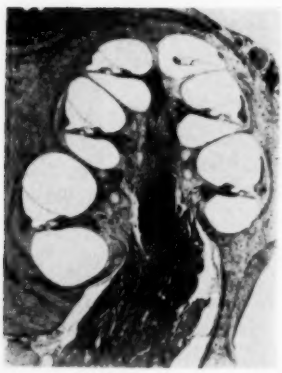


Fig. 3.—Low power view of cochlea (A 496 R/40) of stimulated side showing concavity of Reissner's membrane; endolymphatic compression of Mygind.

middle ear as well as those on the membranous lateral semicircular canal wall following injection of the stellate ganglion (Fig. 4). The patient in this case was under local anesthesia and this may be of significance.[32]

In contradistinction Rambo et al.[17] denervated the sympathetic supply of the monkey's inner ear and did not find a change in the calibre in the labyrinthine vessels when examined microscopically in varying periods up to 60 days later. The same result followed similar treatment of the parasympathetic supply. Rambo et al. also exposed the membranous lateral canal and noted that following sympthectomy and application of pilocarpine (to assure over-activity of the parasympathetics) there was an immediate increased calibre of the vessels of the middle ear drum but no change in the membranous labyrinth. The monkeys were under general anesthesia. This, then, is a variance with the work of Passe. In the two instances the anesthesia was different, local vs. general.

Using dogs under barbiturate anesthesia Strong[32] obtained rather similar results to Rambo et al. After exposing the lateral semicircular canal, a fenestra was created so as to bring into view one or two blood vessels lying on the membranous labyrinth. In six animals stimulation of the stellate ganglion or vagosympathetic trunk failed

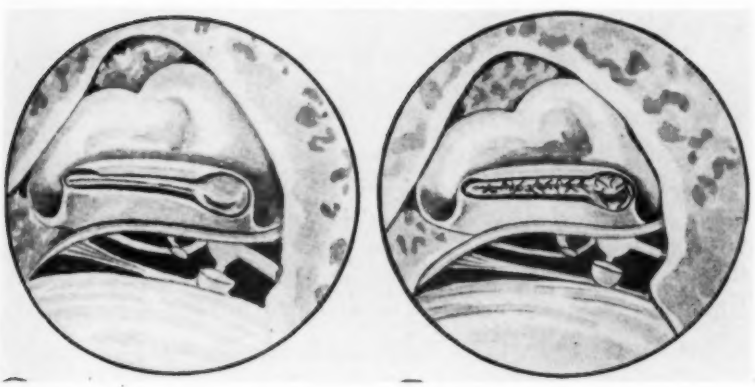


Fig. 4.—*Left*: normal appearance of membranous canal and utricle as seen on fenestration of lateral semicircular canal for otosclerosis.

Right: gross dilatation of blood vessels of supporting trabeculae and of surface of membranous canal as seen after novocain block of stellate ganglion during fenestration under local anesthesia (without adrenaline).

to change the calibre of the vessels or the rate of blood flow. Blocking the stellate ganglion by procaine or surgical section also failed to influence the calibre of the vessel of the membranous labyrinth. Intravenous use of vasoconstriction and vasodilation also did not change the labyrinthine vessels though extra labyrinthine vascular changes were apparent.

And then there is the confusing observation of Mygind and Falbe-Hansen[18] which suggests that the vessels of the labyrinth, like the coronary vessels, may react in a paradoxical way. These observers say the labyrinth of the guinea pig shows vasoconstriction after injection of histamine, vasodilation after adrenalin.

There have been several reports of the effect on vertigo following surgery of the sympathetic nervous system. The work of Garnett-Passe, which was reported posthumously,[33] showed an approximately 60 per cent favorable response to this type of therapy. In 1954 Lewis[34] reported on 12 cases of incapacitating Ménière's disease. In 7 out of the 17 he obtained satisfactory control of the attacks. These patients were subjected either to section of the sympathetic roots of

T2, T3, and T4 or to stellate ganglionectomy. The approximately 41 per cent success obtained by Lewis is at variance with that of Garnett-Passe.

At the Lahey Clinic three cases have been reported[19] in which the stellate ganglion was excised and the vertebral artery ligated. All developed Horner's syndrome. After a period of two years relief has been gained in two of the three.

Strong[32] reported on nine cases subjected to dorsal sympathectomy. In six out of the nine cases the patients consider the vertigo sufficiently less following operation to be pleased with the results. In only one was the patient free of the necessity to take medication, however. These cases include those of Leighton Johnson.

Personally I have had little experience regarding sympathectomy or stellate ganglion block. For the time being I have decided both must be considered experimental procedures and perhaps considered for those cases of intractable Ménière's disease in which the opposite ear is quite deaf making a labyrinthotomy impossible.

Alcohol injections into the oval window (Wright 1938) and the lateral semicircular canal (Mollison 1939) have been largely abandoned because of danger to the VII cranial nerve.

Likewise surgery of the chorda tympani to relieve vertigo as reported by Rosen[21] has not been borne out to my knowledge. Eliasson[22] specifically has questioned Rosen. Sectioning of the chorda tympani has not been useful in my experience.

The last subject which I propose to discuss is the destruction of the labyrinth. In those cases of Ménière's disease in which the vertigo is severe and intractable but the tinnitus is negligible and the hearing good destruction of the vestibular but preservation of the cochlear labyrinth would be desirable. Fleet[24] and Portmann[25] believe that incision of the saccus endolymphaticus accomplishes this in some cases. Fleet reports that of 73 operations in which the saccus was opened for Ménière's disease his results were as follows: postoperative deaths 2, immediate failures 10, wrong diagnosis 1, "perfect results" 19, and improved 43. He concludes that it was a worthwhile operation to do as a preliminary to a destructive procedure.

The technique is simply to elevate the dura mater from the posterior surface of the petrous pyramid. The saccus is found 1-1½ cm distant from the lateral sinus. It is incised and theoretically the

excess pressure of the endolymphatic fluid is thus reduced. Lindsay[26] found that in the monkey the surgical obliteration of the endolymphatic sac and duct was not followed by dilation of the endolymphatic system but on the contrary by prompt healing with closure of the defect in the bone. Fleet[1] believes this failure was due to the monkeys being normal animals with normal endolymphatic ducts. In any event the success of the operation depends on the incised saccus staying open so that excessive accumulation of fluid has a portal of egress.

Altman[27] concluded a few years ago, after reviewing the literature and his cases, that the results of Portmann's operation were not impressive but were worth trying in all cases with serviceable hearing in the affected ear. I would add that the degree of tinnitus should be taken into consideration too. There seems to be little use in preserving hearing in a patient in whom the tinnitus is disabling. It would seem wiser to choose in unilateral cases with good hearing in the opposite ear a different operation and eradicate the tinnitus and the hearing along with the vertigo in the affected ear.

Putnam[28] reported in 1938 five operations on two patients suffering from intractable vertigo. In both the vertigo was relieved and the hearing retained. His method was to selectively destroy the vestibular labyrinth by a drill and electrocoagulation of the superior semicircular canal. His approach was by way of the anterior surface of the petrous ridge via a small subtemporal decompression. Follow-up studies were not given.

Most attention nowadays is directed to the destruction of the labyrinth through an end- or postaural approach. Of the many variations I shall speak principally of three methods: the so-called Lempert decompression,[29] the Day operation,[30] and the Cawthorne operation.[31]

Through a transtympanic approach Lempert cuts the incus near the junction with the head of the stapes and removes the stapes. The rough window membrane is destroyed with a burr. The end picture before replacing the drum is shown in Figure 5. Of the 15 cases reported all were relieved of the vertigo while the tinnitus remained.

Day exposes the lateral semicircular canal through a partial simple mastoidectomy. With a burr just above the medial to the short process of the incus the lateral canal is opened and the membranous labyrinth removed with a dental broach. The needle electrode is then inserted and the contents coagulated, care being taken to avoid the facial nerve. Injury to the facial nerve occurs occasionally. He

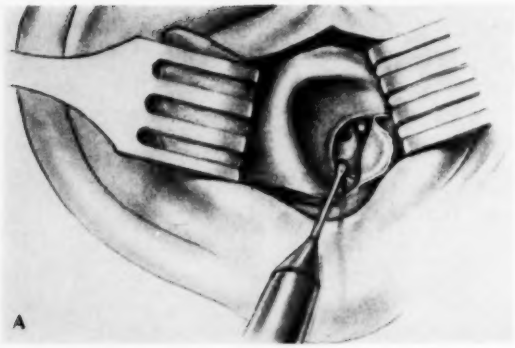


Fig. 5.—Lempert decompression. The stapes has been removed and the round window is being destroyed by a burr.

reports that in 20 out of 21 cases the tinnitus remained in some degree following the operation although the vertigo was eradicated in all. Unfortunately the length of time of followup was not given.

Cawthorne opens the bony external semicircular canal with a dental drill. The membranous canal is then removed by avulsion. Cawthorne and Hewlett[20] reported that this procedure never failed to achieve total loss of both vestibular and cochlear function. In 288 cases reported by them none have ever had a recurrence in the operated ear. There was a transient facial paralysis in one but otherwise no significant postoperative complication.

Lindsay and Siedentop[8] have recently reviewed the various methods, including Cawthorne's, and have given their experience with many of them. While many of the techniques tried were successful in relieving the vertigo they were less so in relieving the tinnitus. To ensure the eradication of both symptoms they suggest creating a window in both the lateral and superior canals and enlarging these so as to form a single broad opening leading into the vestibule. The membranous structures in the vestibule and both canals are ablated by hooks and forceps. Bone chips are then placed in the vestibule to promote osteogeneses. This is the method I currently am interested in and in a short experience seems valuable.

SUMMARY AND CONCLUSIONS

Surgery of the suppurative labyrinth, fortunately, is now of infrequent occurrence and I find it difficult to draw conclusions until more experience is reported.

The treatment of nonsuppurative labyrinthine disharmony by surgical attention to the autonomic nervous system is not yet on a firm basis and, in my opinion, should not be recommended except on an experimental basis. With additional knowledge this avenue of approach may offer a great deal. Its field of greatest usefulness will be in those cases where it is necessary to preserve the hearing.

Differential section of the VIII cranial nerve and surgical destruction of the labyrinth, according to the available information, have been equally successful in eradicating the vertigo of Ménière's disease. The former, however, is preferable if it is important to retain the hearing insofar as possible. It must always be kept in mind, in this regard, that the hearing which you are trying to preserve may be so distorted and the tinnitus so bothersome that subjecting the patient to a long, difficult procedure such as differential section of the VIII nerve may not be in the best interests of the patient.

It seems clear that if the case under consideration is unilateral and the hearing poor in the affected ear either coagulation or avulsion of the membranous labyrinth is preferable to differential section of the VIII nerve. Personally I prefer avulsion.

Only very rarely can a patient with bilateral destructive labyrinthotomy walk unaided, drive a car, etc. Such a procedure is rarely justified.

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VI

RHINOSCLEROMA OBSERVED FOR 23 YEARS

REPORT OF A CASE

FRED W. DIXON, M.D.

CLEVELAND, O.

The patient, L.F., male, was admitted to St. Alexis Hospital, Cleveland, Ohio, in June 1934 in marked respiratory distress. There was a history of nasal crusting and difficulty in breathing while working as a stonemason in Yugoslavia before emigrating to America in 1902. In 1904 and again in 1914 a tracheotomy was performed for difficulty in breathing. This was diagnosed at that time as tuberculosis or syphilis.

Due to cyanosis on admission, an emergency tracheotomy was done. The trachea was located without difficulty, but was so narrowed that a low tracheotomy was necessary to allow a tube to be placed. Subsequently, the upper trachea was dilated until a 7 mm bronchoscope could be passed, at which time the tracheotomy wound was allowed to close.

Biopsy of the material from the nose and trachea showed a typical picture of rhinoscleroma. Approximately every three years there was a recurrence of the obstruction in the nose and trachea. This was removed and followed by roentgen ray therapy. On June 21, 1957, granulation tissue was removed from the nose, posterior surface of the epiglottis and trachea. This was followed by roentgen ray therapy with relief of his breathing difficulty. The pathological picture continuously showed foam cells and Russell's bodies. On December 24, 1957, he suffered several small cerebral hemorrhages and expired at the age of 80.

Autopsy showed a patulous fibrotic trachea smaller than usual, but with no recurrence of the growth. The bronchi had never been invaded.

This man had raised a family, led a normal life and was never greatly inconvenienced by the disease.

1027 ROSE BUILDING

VII

A REPORT OF TWO NORMAL HEARING STUDIES

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The need for an international standard reference level for normal hearing is an accepted fact. Recent criticisms of the present American standard reference level have cast considerable doubt on its validity and, therefore, its acceptance as an international standard. Most of the past criticisms have been in the form of complaints from various otologists who have reported that they are continually confronted with many young persons whose auditory threshold is consistently lower than the American reference level. These observations recently were emphasized by Dadson and King[1] and Wheeler and Dickson[2] of the National Physical Laboratories and the Central Medical Establishment of the Royal Air Force in Great Britain.

An examination of the United States Public Health Service data,[3] the World's Fair[4] and San Diego Fair data,[5] the Sivian and White,[6] and Bell Telephone Laboratories[7] data permits us to separate these studies into two general groups, 1) mass surveys and 2) specific laboratory studies. When the data are looked at with this in mind the survey results resemble the present American standard rather closely and the laboratory data are in rather close agreement with those recently reported by the British. There are many points of difference between all of these studies, and one logically may argue that different equipment, test environments, psycho-acoustic techniques, and statistical treatment will contribute to discrepancies in the final data. However, when all of these facts are taken into account the differences between the studies in each of the two classes are negligible, and the grouping remains the same. It is apparent that more investigation is necessary to determine the cause of this difference between these two types of studies. With this in mind the Research Staff of the Subcommittee on Noise in Industry conducted two hearing studies at the Wisconsin State Fair in August of 1954 and 1955.

Read at the Sixth International Congress of Otolaryngology, Washington, D. C., May 1957.

From the Research Center, Subcommittee on Noise in Industry.

Both surveys were made possible by the co-operation of the Wisconsin Manufacturers Association, the State and County Medical Societies and the State Teachers College of Milwaukee. The following is a report of the data relating to the normal hearing reference level.

REPORT OF NORMAL HEARING DATA FROM THE 1954 WISCONSIN STATE FAIR SURVEY

In 1954, the auditory thresholds of 3515 persons between the ages of 12 and 79 were tested with pure tones and speech under excellent environmental conditions. The auditory thresholds for pure tones of each subject were measured by air conduction in 5 db steps with a standard diagnostic audiometer. The audiometers were supplied by the Maico Company of Minneapolis, Minn. All the audiometers were calibrated by the manufacturer prior to the survey, several times during the project by the staff and after completion of the project by an independent laboratory. Each person was tested only once by the conventional manual procedure, which is a modification of the method of limits. The frequencies tested were 500, 1000, 1500, 2000, 3000, 4000 and 6000 cps. The binaural earphones used were PDR-10's made by Permoflux with standard MX 41/AR cushions and the usual head-band. Each subject was examined and questioned by an otologist, and all indications of pathology and historically significant items were recorded. The ambient noise levels in the test booths were well within those being considered by the American Standards Association as suitable for audiometry rooms.

The sample chosen from these records for consideration as a normal hearing group consisted of the records of 202 persons (72 men and 130 women) between 18-24 years of age. No one in this restricted group had any evidence of auditory pathology or significant noise exposure so far as could be determined by an otological history and examination.

Table I indicates the pure tone threshold values in db re American reference level. The data obtained by Beasley from the United States Public Health Service survey used for the present American normal hearing reference level, are compared to those from the 1954 Wisconsin State Fair normal hearing group in Table II. The values are given in sound pressure level in a National Bureau of Standards 9A coupler.

TABLE I

MEDIAN HEARING LEVEL FOR MEN AND WOMEN
IN A RESTRICTED 18-24 YEAR OLD GROUP
Wisconsin State Fair
1954

	MALE		FEMALE		MALE AND FEMALE	
FREQUENCY	RIGHT	LEFT	RIGHT	LEFT	RIGHT	LEFT
500	—0.8	1.7	—0.6	—0.7	—0.7	0.2
1000	1.0	1.8	0.5	0.0	0.6	0.6
1500	0.7	1.0	0.9	0.4	0.8	0.6
2000	0.8	2.2	1.9	0.9	1.5	1.4
3000	2.8	5.6	3.0	3.8	2.9	4.6
4000	4.7	7.5	5.2	5.6	5.0	6.2
6000	9.5	13.6	8.6	9.5	8.9	11.0
Number	72		130		202	

No persons were included who had any of the following:

1. Factory work
2. Noise exposure
3. Military service
4. Trouble with ears
5. Perforations of drumhead
6. Discharge from ears

TABLE II

COMPARISON OF PRESENT AMERICAN NORMAL HEARING REFERENCE LEVEL AND 1954 WISCONSIN STATE FAIR RESTRICTED 18-24 YEAR OLD GROUP DATA. SPL IN A NBS 9A COUPLER

CPS	AMERICAN NORMAL U.S.P.H.S. DATA[1]	1954 W.S.F. 18-24 YEAR OLDS DATA[2]
125	52.4	----
250	38.8	----
500	24.5	24.1
1000	16.5	15.6
2000	17.1	16.9
4000	14.5	17.8
8000	28.3	----

[1] 705A Western Electric earphone values reduced to pressures obtained in PDR-8 earphones by National Bureau of Standards.

[2] PDR-10 Permoflux earphone (Medians). (Both ears).

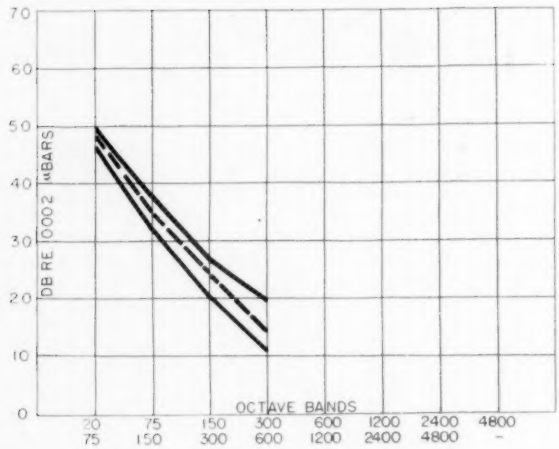

Fig. 1.—Average, maximum and minimum sound levels measured in the test booths used at the 1954 and 1955 Wisconsin State Fair hearing surveys. Readings beyond the 300-600 cps band were too low to be recorded.

REPORT OF NORMAL HEARING DATA FROM THE 1955 WISCONSIN STATE FAIR STUDY

In August 1955 at the Wisconsin State Fair, the research staff of the Subcommittee on Noise in Industry performed a carefully controlled study of the auditory threshold in 122 specially selected "fair-goers" between 18 and 24 years of age. Each subject was questioned regarding familial hearing loss, past and present history of otological disease and noise-exposure. Only those who were completely normal to physical examination, who gave no history of otological disease in themselves or their families, and who had little or no exposure to high level noise, were chosen. Fifty-seven males and 65 females completed the test.

After careful instruction regarding the test, each subject was seated in a specially prepared sound-treated booth with ambient noise levels as indicated in Figure 1. The prefabricated audiometry booths were furnished by the Industrial Acoustics Company of New York, N. Y. The booth was air-conditioned and kept at a relatively constant comfortable temperature. No one but the subject remained in the

booth once a test had been started. All the equipment was located outside the test booth, and visual contact was maintained through a large glass window. The subject signalled the operator by raising his or her finger whenever, and as long as, each stimulus tone was heard. When the tone could not be heard, the finger was lowered. Each subject first was tested in each ear with nine pure tones (125, 250, 500, 1000, 2000, 3000, 4000, 6000 and 8000 cps) at 5 db steps. Following this, one ear was selected by a counterbalanced system and was tested twice at the same frequencies in 2½ db steps. The responses were not recorded until the threshold had been crossed at least three times in each direction. In order to avoid biasing the operator's decision in favor of the American Medical Association "zero," one audiometer was equipped with a recording device which made it unnecessary to read the attenuator dial. This device was built by J. J. Cantor of the Surgical Mechanical Research Company, Los Angeles, Calif. The threshold values were printed on a paper roll by depressing a foot pedal. This method of recording was used on 47 of the subjects. The thresholds of the remaining subjects were recorded by reading the dial as in conventional audiometry. The above procedure required from 30 to 45 minutes to complete. Sixty-one right ears and 61 left ears were tested in this manner.

Two modified commercially available audiometers were used. The attenuator in each was adjusted to attenuate in 2½ db steps to 20 db below the present American reference level for normal hearing. Telephonic (TDH-39), dynamic type earphones were used. Standard MX 41/AR cushions coupled the earphones to the ears and a conventional headband held them in place. Measurements indicated that the earphones exerted, approximately, 1000 gms of pressure against each ear when the headband was properly situated.

Both audiometers were carefully calibrated with a National Bureau of Standards 6 cc, 9A artificial ear. Voltage readings also were taken at the earphone terminals at 60 and 70 db re 0.0002 dyne/sq. cm. Each instrument was checked to the limits of the attenuator by a special technique designed for the purpose. All of these calibrations were made in an independent laboratory prior to, and subsequent to, the actual study. The audiometers were calibrated at the survey site in a National Bureau of Standards 9A coupler several times each day during the ten day period.

Table III presents the threshold values in db re American reference level, obtained in the selected group at the 1955 Wis-

TABLE III

THRESHOLDS FOR THE "SELECTED" 18-24 YEAR OLD GROUP IN THE
1955 WISCONSIN STATE FAIR HEARING SURVEY
(DB RE AMERICAN AUDIOMETER ZERO)
N = 122 (61-R; 61-L)

	EAR														
	RIGHT					LEFT					RIGHT AND LEFT				
FREQUENCY	MODE	MDN.	MEAN$_r$	S.D.$_r$	N$_r$	MODE	MDN.	MEAN$_r$	S.D.$_r$	N$_r$	MODE	MDN.	MEAN$_r$	S.D.$_r$	N$_r$
125*	—5.0	—3.8	—3.1	4.5	38	—5.0	—3.2	—2.8	5.0	38	—5.0	—3.6	—3.0	4.7	76
250	—12.5	—12.0	—11.2	5.0	60	—13.8	—13.0	—12.9	5.4	59	—12.5	—12.4	—12.0	5.3	119
500	—7.5	—11.6	—11.0	5.4	61	—17.5	—12.2	—12.6	5.1	58	—17.5	—11.9	—12.2	4.7	115
1000	—10.0	—9.4	—9.3	4.9	61	—10.0	—9.6	—9.8	5.2	57	—10.0	—9.5	—9.5	5.0	118
2000*	—12.5	—9.6	—9.2	3.6	60	—10.0	—8.2	—8.2	6.4	59	—10.0	—9.1	—8.6	5.3	120
3000	—7.5	—6.1	—4.9	5.3	57	—2.5	—3.2	—3.1	5.2	60	—7.5	—4.6	—4.1	5.3	116
4000	—5.0	—4.8	—5.0	6.0	58	2.5	—1.4	—0.4	6.1	61	—5.0	—3.4	—3.2	5.9	116
6000	—6.2	—1.9	—3.1	6.6	52	2.5	—2.4	—1.0	7.4	59	—5.0	—2.2	—2.6	6.6	111
8000	—7.5	—4.6	—4.6	5.4	54	—10.0	—7.2	—8.2	6.2	52	—7.5	—5.7	—6.2	6.2	107

* N at 125 cps was 78 (39-R; 39-L), and at 2000 cps was 121 (61-R; 60-L).
The subscript r refers to the restricted sample of ± 15 db around the mode.

consin State Fair. The data are tabulated as modal, median, and restricted mean (mean$_r$) values. The restricted mean was obtained by limiting the data to ± 15 db around the mode. The standard deviations and N's were derived from the restricted group. The following examples illustrate the distribution of the threshold values. Ninety-one per cent of the threshold values at 500 cps fell within a range of 15 db, 89 per cent at 1000 cps fell within 20 db and 82 per cent at 4000 cps fell within 16.5 db.

COMMENT

When Tables I and III are compared, the difference between a survey study and a laboratory study still is very much in evidence. Both of these studies were controlled as carefully as possible in every way. They were conducted for the purpose of obtaining additional data on the problem of a standard normal hearing reference level, and even the severest critic would find it hard to eliminate any of the data on the basis of environment, calibration, technique, or other controllable contributing factors to higher thresholds. Every possible safeguard against artifacts in the data was carefully considered and was applied wherever practicable. It is evident that the explanation of the differences noted in Tables I and III is related to factors which have no relation to the test environment, instrument calibration, technical personnel, or pathological causes of hearing loss. The following is a discussion of these differences and some of the possible contributing factors.

The previously mentioned new normal hearing reference zero was proposed in 1952 by the National Physical Laboratories of Great Britain, on the basis of auditory threshold determination made on two groups of listeners totaling 611 persons between 18 and 25 years of age. The thresholds in one group of 99 subjects were reported by Dadson and King,[8] and those from the remaining 512 subjects were reported by Wheeler and Dickson.[9] The agreement between Dadson and King and Wheeler and Dickson is excellent, and the Dadson and King data will be used for comparative purposes in this report. Table IV gives a comparison of the Dadson and King data and the 1955 Wisconsin State Fair data in modal, median and mean values, expressed as sound pressure level. The values in these studies were very close at all frequencies except 3000, 6000, and 8000 cps. The British sound pressure levels were obtained with a 4026A type earphone in a British type 3 cc artificial ear coupler.[10] The 1955 Wisconsin State Fair

TABLE IV

COMPARISON OF 1955 WISCONSIN STATE FAIR[1] AND DADSON AND KING[2] AUDITORY THRESHOLD DATA

SPL IN ARTIFICIAL EAR

(DB RE 0.0002 DYNE/SQ. CM.)

	MODE		MEDIAN		MEAN		S D (DB HEARING LEVEL)	
CPS	1955 WSF	DADSON-KING	1955 WSF	DADSON-KING	1955 WSF	DADSON-KING	1955 WSF	DADSON-KING
125	46.2	44.5	47.6	45.5	48.6	45.5	5.3	6.9
250	26.2	28.5	26.3	28.0	27.2	28.0	6.1	7.3
500	6.9	11.5	12.5	12.0	13.1	12.5	6.0	6.5
1000	7.4	6.0	7.9	5.5	8.6	5.5	6.2	5.7
2000	9.1	9.5	10.0	11.0	10.7	10.5	5.5	6.1
3000	9.5	6.0	12.4	6.5	13.8	7.0	6.5	5.9
4000	8.6	8.5	10.2	8.5	11.4	9.5	7.2	6.9
6000	18.0	9.0	20.8	10.5	22.0	10.5	8.3	9.1
8000	14.3	9.5	16.1	9.0	17.8	9.0	8.4	8.7

[1] Telephonics Earphones (TDH-39) (NBS 9A Coupler)

[2] British type Earphones (4026A) (British 3 cc. Coupler)

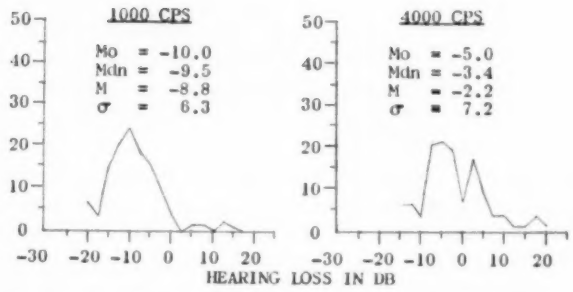

Fig. 2.—Distribution of threshold values at 1000 and 4000 cps in a selected sample of males and females 18-24 years of age. (R & L Ears, N = 122) Wisconsin State Fair 1955.

values were obtained with a Telephonic TDH-39 earphone in a National Bureau of Standards 6 cc 9A artificial ear coupler.

When the mode, median and means are compared, there are only minor differences that may be a function of the distribution of the hearing levels in the sample. The distribution values in the 1955 Wisconsin State Fair study shown in Figure 2 readily explain why. With so little skewing one would not expect great differences in these measures of central tendency. This can be seen in Table III where the mode and median are compared with a restricted mean. There is very little choice, particularly between the median and restricted mean (mean$_r$). The slight differences noted between the modal values and those shown by the median and restricted mean are probably due to the instability of the mode. When the distribution of the data is relatively flat or tends toward bimodality, the exact mode is difficult to identify accurately.

With the agreement so close at all other frequencies, it is reasonable to assume that differences in earphones may explain the relatively minor discrepancies in some of the frequencies. Here, then, are two studies carried out in much the same way as regards purpose, environment, technique, and subject selection. The only real difference is in the equipment used, notably the earphones and artificial ears. Despite this, the values obtained are essentially equivalent.

It is apparent from the 1954 Wisconsin State Fair study that even in a well-controlled survey the threshold values remain very

much the same as those used in the present American standard reference level. Probably the most significant finding in the 1954 Wisconsin State Fair survey is the relatively good agreement with the present American reference level and the persistence of the difference previously found between survey and laboratory studies.

When a comparison is made of the 1954 and 1955 Wisconsin State Fair data, there appears to be a distinct separation between them, of the same magnitude, as was found between previous survey and laboratory studies. When all the available studies are examined, the small variances among the individual similar type studies are negligible, and one is left with the problem of explaining the obvious separation of the data into two major classes. When these two classes are examined, the distinguishing factors seem to be related to the procedural variations inherent in the type of study.

If one takes a good look at these two types of studies it is easy to see several very striking differences in procedure. The survey is designed to produce a large amount of data in a relatively short time. And although a reasonable amount of care is taken to obtain good data, within the allotted time, no real effort is made to insure good test reliability. The subject is tested only once in each ear at 5 db steps with tones which he probably has never heard before. He is hurried into a sound treated, small-sized space and rapidly told in a few words that he must raise his finger when he hears some faint music-like tones. By the time he has realized what it is all about, the test is over, and he is asked to make room for the next subject. Further, the technician, who is reasonably well trained, records only one judgment after roughly bracketing the threshold area. No attempt is made to record multiple responses to provide a mean threshold value. This is accomplished by a rapid mental process while the test is proceeding. On the other hand, the laboratory study is designed to produce a relatively small amount of data with little or no regard for time. The subject is carefully instructed in the test procedure and allowed to ask questions if he desires to do so. He is then tested at least twice and sometimes three times. The threshold is crossed in both directions at least three times in one or two db steps and a careful record is made of all responses to allow for statistical treatment when the absolute threshold is calculated. When such variations in procedure are considered, it seems quite logical to assume that the consistent difference recorded between survey and laboratory studies more likely is due to the test-subject relationship than any other factor.

FACTORS THAT MIGHT INFLUENCE THE DATA FROM EITHER SURVEY OR LABORATORY STUDIES

The available reports of studies on the normal hearing reference level devote a great deal of space to discussing the effects of variations in equipment used for psychophysical, and purely physical acoustical measurements. Much has been said about auditory measurements as derived by probe tubes, artificial ear coupler, earphones and loudness balancing techniques; but none of the reports has included any data on other variables usually referred to as biases. It is generally not expedient to control such variables and the researcher is left wondering just what have been the effects of these biases on the data.

In a report from Working Group 2 of the Armed Forces-National Research Council Committee on Hearing and Bio-Acoustics outlining a suggested procedure for obtaining normal hearing data, several of these biases were mentioned and were considered important. In an effort to obtain data on some of these possible biases, the Wisconsin State Fair studies were analyzed for the effects of: 1) the subject's opinion of his own hearing, 2) education, 3) practice effects, 4) pathology, and 5) the effects of variables associated with the technician.

1. *Representativeness of the Sample.* It always is difficult to be reasonably sure that a sample chosen for study is representative of a total population. In any hearing survey where no rigid selection of the subjects is planned, one always wonders how many of the volunteers respond because they believe they have poor hearing and are interested in a test on this account. In order to test for this possible bias, each subject was asked whether he thought his hearing was good, fair, or poor. In the 1954 Wisconsin State Fair studies, 18-24 year old group, 0.6 of 1 per cent thought their hearing was poor. On the basis of this it seems quite safe to assume that this bias was not operating to produce a higher threshold in this group.

2. *Education.* Education, and its relevant effects on the ability of a subject to make more accurate observational responses, has been thought to cause artifacts in threshold measurements of any kind. Each subject, in both Wisconsin State Fair studies, was asked how many grades in school he had completed. On the strength of this information, a comparison between the thresholds of those persons with less than a ninth grade education, and of those with a ninth grade or more education, in the 1954 study, is shown in Table V.

TABLE V
TWENTY-FIFTH PERCENTILE, MEDIAN, AND SEVENTY-FIFTH PERCENTILE HEARING LEVEL FOR PERSONS WITH LESS THAN A 9TH GRADE EDUCATION AND FOR PERSONS WITH A 9TH GRADE EDUCATION OR MORE

			AGE							
			30 - 39 YEARS EDUCATION		40 - 49 YEARS EDUCATION		50 - 59 YEARS EDUCATION		60 - 69 YEARS EDUCATION	
SEX	FREQUENCY	EAR	0-8	9 +	0-8	9 +	0-8	9 +	0-8	9 +
Male	1000	R	3.9	0.1	4.2	2.7	8.1	3.7	14.6	6.2
			8.4	4.8	10.6	8.1	16.3	8.6	27.3	15.4
			14.6	11.2	26.6	19.4	38.4	22.5	46.1	35.3
		L	5.1	0.4	5.5	2.8	8.7	3.5	14.0	8.6
			9.7	5.1	11.9	9.2	18.2	9.3	27.8	20.2
			15.6	11.7	28.4	21.1	38.0	25.0	46.4	39.6
	4000	R	23.8	10.0	28.4	17.6	41.6	24.5	57.6	35.9
			37.0	23.6	46.8	34.1	56.4	46.8	64.8	55.0
			50.6	45.8	62.5	55.8	71.0	62.5	74.9	72.8
		L	31.9	13.8	32.5	19.8	43.9	30.7	55.4	44.8
			44.0	29.6	53.0	36.5	57.1	49.8	65.3	59.0
			54.8	52.8	70.4	56.4	71.8	63.0	77.4	75.3
	Number tested		35	311	80	221	139	180	113	83
Female	1000	R	—0.3	—1.0	—1.1	1.3	6.0	2.0	11.6	7.2
			5.0	3.6	6.0	6.4	12.6	8.5	21.6	16.2
			13.8	9.9	16.6	17.7	21.2	17.6	36.4	35.4
		L	0.7	—1.5	0.8	0.7	4.3	2.0	9.9	7.1
			6.6	3.4	5.2	5.8	10.4	7.7	19.2	15.0
			14.4	9.6	12.7	15.6	23.6	18.2	36.1	29.7
	4000	R	4.6	4.3	8.2	6.5	17.9	12.3	25.8	21.6
			10.8	10.2	15.4	13.5	25.4	20.5	38.0	32.9
			23.4	19.0	26.0	24.6	42.8	33.4	67.1	49.4
		L	9.6	6.2	10.3	9.4	16.5	14.0	24.3	23.3
			15.8	12.5	17.3	16.0	29.1	23.0	39.4	37.8
			23.8	21.9	27.7	26.8	44.0	36.4	59.7	53.2
	Number tested		30	282	75	285	115	199	85	69

It would be difficult to draw any firm conclusions from this comparison for it must be presumed that there are inherent biases here that would favor the more educated group. For example, one logically might assume that the group with more education would include a greater number of mentally alert and trained persons who would, therefore, make better observations. Such differences as those found in Table V must be related to many influences that may be grouped under the general term "education." However, it seems reasonably safe to conclude that whatever education signifies, more education seems to be related to lower auditory thresholds. Various analyses of these data, based on the amount of education and its relation to the auditory threshold, place the maximum difference between the two groups as shown in Table V. When comparisons were made between other groupings the differences were not as significant.

3. *Practice.* There is no doubt in the minds of anyone that there is a "practice effect" in audiometry. Just when, and for how long, this operates is not known, however. Both Wisconsin State Fair projects presented opportunities to study this effect. In 1954, the order of frequencies tested followed that used in clinical audiometry. Each test was started at 1000 cps, proceeded through 1500, 2000, 3000, 4000 and 6000 cps, and returned to 1000 cps again, finally finishing at 500 cps. When the data were to be analyzed, the choice had to be made of which response at 1000 cps to use. In order to make this choice, 200 randomly selected records were chosen and compared on the basis of the first and second response at 1000 cps. The comparison showed a lower threshold for the second test at 1000 cps in the first ear tested, but little, if any, difference for the second ear tested. The comparison did not, however, determine if this apparent practice effect carried over into any of the succeeding frequencies. It did show that the "effect" had disappeared before the first test at 1000 cps in the second ear.

When a comparison is made between the first and second ear tested in 1954 at 1000 and 4000 cps the results obtained are shown in Table VI. In order to obtain more data on the effect of repeat tests, approximately 300 of the persons tested in 1954 were asked to return in 1955 to be retested. One hundred and thirty-six of these individuals responded. Table VII shows the comparisons made from these two tests on the same persons under similar testing conditions one year apart. Notice that the correlations between these two tests on the same individuals are high, but that the group values show lower thresholds for the tests made in 1955. The difference at 4000 cps

TABLE VI

MEDIAN HEARING LEVEL FOR THE FIRST AND SECOND EAR TESTED FOR MALES 20-29 YEARS OF AGE (1954)

FREQUENCY		FIRST EAR	SECOND EAR
1000		3.7	3.1
4000		14.5	13.0
Speech Reception		10.6	10.2
Number Tested	R	156	163
	L	163	156

Differences not significant at the 5% level.

TABLE VII

AVERAGE THRESHOLDS OBTAINED IN THE 1954 WISCONSIN STATE FAIR HEARING SURVEY AND IN THE 1955 WISCONSIN STATE FAIR HEARING SURVEY FOR THOSE TESTED AT BOTH FAIRS (N = 136)

FREQUENCY	EAR	MEASURE	YEAR 1954	YEAR 1955	CORRELATION BETWEEN THRESHOLDS OBTAINED IN 1954 AND 1955
1000	R	Mean	9.5	8.2	.87
		Median	6.1	4.6	
	L	Mean	9.7	8.3	.92
		Median	5.8	4.4	
4000	R	Mean	23.1	18.4	.89
		Median	14.6	11.8	
	L	Mean	25.5	19.8	.93
		Median	18.4	13.5	

particularly is noteworthy. One reason for this improvement might be the effect of practice. If this is so, however, the practice effect has persisted over a year. It is rather hard to believe that a practice effect would persist for so long after only one audiogram.

When the 1955 Wisconsin State Fair normal hearing data are analyzed for practice effect between the first and second tests on the

TABLE VIII

AVERAGE HEARING LEVEL OBTAINED WITH 5 AND 2.5 DB STEPS FOR THE "SELECTED" GROUP AT THE 1955 WISCONSIN STATE FAIR HEARING SURVEY
(N = 122)

FREQUENCY	MEAN HEARING LEVEL 5 DB STEP	2.5 DB STEP	MEDIAN HEARING LEVEL 5 DB STEP	2.5 DB STEP
125*	2.8	—2.6	3.6	—3.6
250	—6.4	—11.5	—7.9	—12.4
500	—7.1	—11.3	—7.3	—11.9
1000	—5.6	—8.8	—7.5	—9.5
2000	—4.4	—8.4	—4.6	—9.1
3000	1.6	—3.2	1.7	—4.6
4000	—1.0	—2.2	—1.8	—3.4
6000	3.5	—1.0	1.8	—2.2
8000	1.5	—4.0	1.1	—5.7

* N at 125 cps = 78

same ear, Table VIII is the result. Notice that the second threshold determination varies from two to six db lower than the first. However, since the first threshold determination was made in 5 db steps as compared to 2½ db steps in the second, it is not certain whether this difference is due to practice or to a change in attenuation steps. It will be remembered that the 1955 normal hearing group was tested in both ears at 5 db steps, and then twice in one ear in 2½ db steps. A comparison between the second and third tests on the same ear (2½ db steps) is shown in Table IX and there is no appreciable difference.

TABLE IX

AVERAGE THRESHOLDS OBTAINED FOR THE FIRST AND SECOND ADMINISTRATION OF THE 2.5 DB TEST TO THE "SELECTED" GROUP AT THE 1955 WISCONSIN STATE FAIR HEARING SURVEY
(N = 122)

FREQUENCY	MEAN HEARING LEVEL TEST I	TEST II	MEDIAN HEARING LEVEL TEST I	TEST II
500	—11.4	—11.4	—11.8	—12.0
4000	—2.2	—2.5	—3.8	—3.5

4. *Elimination of Probable Causes of Hearing Loss.* When the 18-24 year old group from the 1954 Wisconsin State Fair study is restricted as shown in Table I, and the resulting threshold values are compared to those obtained from the total group, there is no significant difference. This would seem to indicate, for all practical purposes, the absence of effects of noise-exposure and ear pathology in this age group.

5. *Influence of Technique for Obtaining Threshold.* From time to time, various researchers have wondered just how much the technician was influenced by the knowledge of where zero hearing level is located on the attenuator dial. Table X gives the results as obtained

TABLE X

AVERAGE HEARING LEVEL OBTAINED WITH THE MANUAL AND PRINTER AUDIOMETERS FOR THE "SELECTED" GROUP AT THE 1955 WISCONSIN STATE FAIR HEARING SURVEY
(N = 75 MANUAL; 47 PRINTER)
(2.5 DB STEPS)

FREQUENCY	MEAN HEARING LEVEL MANUAL	MEAN HEARING LEVEL PRINTER	MEDIAN HEARING LEVEL MANUAL	MEDIAN HEARING LEVEL PRINTER
125*	—1.2	—5.1	—1.6	—5.1
250	—10.7	—12.8	—11.5	—13.6
500	—11.0	—11.7	—11.0	—14.2
1000	—8.5	—9.4	—8.3	—10.9
2000	—7.6	—9.7	—9.0	—9.1
3000	—1.9	—5.4	—2.7	—6.7
4000	—2.3	—2.0	—3.3	—3.4
6000	—0.5	—1.8	—1.0	—3.2
8000	—2.1	—6.9	—4.2	—7.0

* N at 125 cps = 50 Manual; 28 Printer

by the printer method, described earlier in this paper. The difference between manual and printer values in Table X was not necessarily due to the knowledge of where zero hearing level was, however. When the data derived from the printer are examined, it is evident that there were many more recorded responses for the same frequency than would result when the technician elicited several responses, but recorded only a single value resulting from a subjective judgment. For

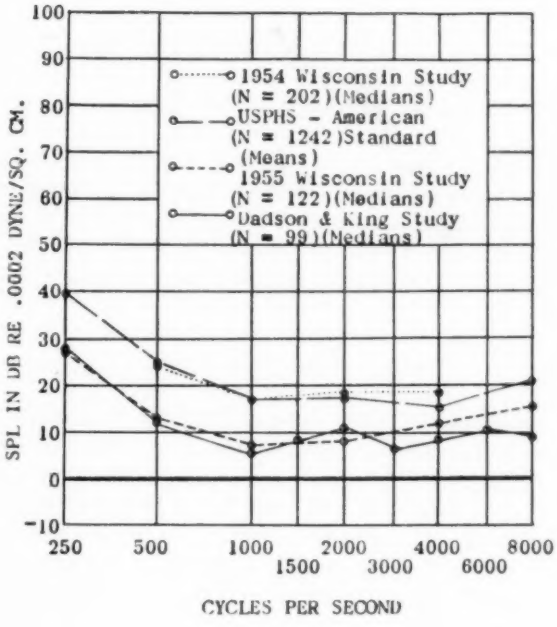

Fig. 3.—Comparison of the auditory thresholds, in sound pressure level, of the 1954 18-24 year old group, the present American normal hearing reference level, the 1955 Wisconsin State Fair 18-24 year old group, and the Dadson and King group.

example, if the subject always responded at minus 2.5 db, and only occasionally at minus 5 db, the technician recorded minus 2.5 db as the threshold whereas the printer method recorded every response, thereby reducing the threshold value by the effect of the minus 5 readings. At least one conclusion can be drawn from these data. Detailed records of all near-threshold responses probably produce lower threshold values than when a single value is recorded as the result of a subjective judgment on the part of the technician.

These data have been included to demonstrate the need for more information regarding such poorly controlled variables. The limited amount of data presented here is sufficient to illustrate some of the possible influences of these biases. They indicate that the variations produced on the auditory threshold by such biases, still are insufficient

in themselves to explain the discrepancy found between survey and laboratory studies of normal hearing.

When all of the data on normal hearing are reviewed, including those from the 1954 and 1955 Wisconsin State Fair studies, there appears to be little doubt that the auditory threshold in a carefully measured and limited group is better than the American standard (Fig. 3) indicates. There is also little doubt that the reason for this apparent discrepancy has little to do with test environment, differences in equipment, technical assistance or statistical treatment of the data. The choice is no longer a technical matter. It is now a philosophical problem and must be decided by common agreement.

SUMMARY AND CONCLUSIONS

1. A determination of the normal threshold of hearing by earphone listening has been made on two groups of persons between the ages of 18 and 24 years. Each group was tested to study the relations of normal hearing to present and proposed standard reference levels for normal hearing. The primary object of the work was to provide additional data which might explain why there are differences in presently reported studies of normal hearing. Variables were controlled wherever possible.

2. The results of these studies agree very well with previous determinations when the conditions of the studies are comparable. The data from the survey conducted in 1954 compare favorably with that from the Public Health, World's Fair, and San Diego surveys. The data from the laboratory study carried out in 1955 compare very well with those from Sivian and White, the Bell Laboratories and the British studies of 1952.

3. Problems of various statistical biases, such as education, practice effect, ear pathology, etc., are discussed in relation to effect on the normal auditory threshold. The data present evidence that auditory threshold data may vary depending on the presence of these variables.

4. It appears that most of the consistent differences found in all of the reported determinations can be explained best by separating the reports into survey and laboratory studies. The agreement among like studies is good except for minor variations which may be ac-

counted for on the basis of differences in technique and sampling. When the survey and laboratory studies are compared, there is a difference in the threshold data with consistently lower values in the latter group.

5. After considering these data, it is felt that the discrepancies noted between the proposed new British reference levels for normal hearing and the present American standard for normal hearing are best explained on the basis of differences in experimental technique inherent in survey and laboratory studies. It appears that the variations found in the equipment used in the U. S. Public Health and the 1954 Wisconsin State Fair Surveys and the Dadson and King and 1955 Wisconsin State Fair studies will not account for the very real discrepancies found between the first two and the last two studies.

6. It is no longer a question of resolving differences in various studies but rather a question of whether survey or laboratory data will be used for establishing a normal hearing reference level.

111 North Bonnie Brae

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VIII

FIBROSING MASTOIDITIS

ITS RELATION TO ADHESIVE OTITIS MEDIA, GRANULOMAS OF THE FACIAL NERVE AND THE CONSERVATION OF HEARING

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Our primary aim in the treatment of middle ear, mastoid and petrosal infections should be to obtain a healthy patient, a nondischarging middle ear, an intact tympanic membrane and normal hearing.

In the pre-antibiotic era the clinical symptoms of ear infections were more dramatic. There was more pain, temperature, leukocytic response, and local findings in proportion. More reliance was placed upon early myringotomy, an antrotomy in infants, a cortical or simple mastoidectomy in other patients, or even a petrosectomy if indicated. Otitis meningitis, lateral sinus thrombosis, or brain abscesses were, of course, more frequent and dreaded complications.

In the antibiotic era many of the acute symptoms which direct our attention to pathology in the temporal bone have been reduced in severity by this type of therapy. There is no doubt that numerous temporal bone infections have been successfully treated by antibiotics. This type of therapy has not only reduced and modified the number of intracranial otitic complications,[1] but has reduced the mortality from otitic complications by about 90 per cent according to Courville.[2] However, when complications do occur, their diagnosis is much more difficult. Unfortunately, also with this era has come a reliance on the use of antibiotic drugs so much so that the basic surgical principles of drainage of suppurative products in an enclosed space has been overlooked or forgotten.

Presented before the Colorado Ear, Nose and Throat Society, September 7, 1957.

From the Ear, Nose and Throat Service, Department of Surgery, Fitzsimons Army Hospital, Denver, Colorado.

Pathological studies and photographs from the Laboratory Service, Colonel Howard A. VanAuken, M.C., Chief of Service.

After using these therapeutic agents, some other factors seem more apparent: 1) the individual's resistance to infection is less often given the opportunity to develop; 2) the bacterial sensitivity of many organisms to antibiotics is becoming more resistant; and 3) the mastoid cells are by development or by mucosal edema and granulations which impede out-flow, unable to drain sufficiently to permit the escape of inflammatory products. While purulent aural discharge may be present or even a subperiosteal abscess developing, there has been a striking reduction of gross purulent discharge from the middle ear and mastoid cells. This has often been referred to as the masking effect of antimicrobial therapy. Attempting to prevent complications of upper respiratory infections by antibiotic therapy seems to add to the complexity of the ear problem. A suppurative discharge may not develop. With the lessening of general symptoms, unless the tympanic membrane is carefully examined and studied, the otological involvement may not be known. With recurrences of respiratory infections, soon the stage is set for either the development of fibrous thickening beneath the epithelium of the middle ear and mastoid, or a more destructive process of the tympanic membrane and ossicles. The latter is usually accompanied by periods of suppuration.

Adhesive otitis media has been, and still is, an important cause of chronic progressive deafness. The cause of these adhesions about the ossicles is infection which has stimulated the formation of granulation tissue. Kobrak[3] has recently stressed that the pathology of non-otosclerotic conduction type deafness is quite varied as to the site, extent and type affecting the middle ear.

After studying a number of these cases which present 1) the history of a present or recurrent middle ear disease, 2) a haziness to cloudiness of the mastoid by x-ray, and 3) a moderate to severe conductive hearing loss, questions arise as to the best method of treatment, if our primary aims are to be obtained. Usually antibiotic therapy had been employed on one, two, six or more occasions. The tympanic membrane is thickened and often scarring or a healed funnel-shaped perforation is present. An existing perforation may contain scanty to profuse secretion. The question is, how much "change" in the mucosal structures of the middle ear is reversible, to permit the restoration of normal hearing and a healed tympanic membrane.

Allergies should be recognized and treated as indicated. However, adequate clinical control of allergic symptoms has not seemed to alter the course of the fibrotic process in the mastoid cells once it has begun.

The tightly packed mastoid cells obscure the radiological picture by replacing the air by granulations. Thus, the mastoid appears and is reported as having antral or periantral sclerosis. In turn, the extent of the mastoid pneumatization is blurred. This may portray a confusing radiological picture.

The pathological picture is one of fibrosis, connective tissue being laid down. The epithelial lining of the mastoid cells may be difficult to find. With this granulomatous reaction in the middle ear space, the epitympanic area, the mastoid antrum and in the mastoid cells, drainage from these various locations is mechanically blocked. This thickening of the lining, which pathologically is fibrous tissue in varying degrees of maturity, seems to be the bodily response to infection as tempered by the individual's resistance, plus the effect of antimicrobial therapy. The cellular infiltrate of the granulations varies in intensity and type. Localized areas of suppuration may be present, adjacent to areas of firm fibrosis, hemorrhage or cholesteatomatous crystals.

This pathological picture coupled with the variability of the extent, size and shape of the mastoid cells, defects or near defects in the bony wall of the fallopian canal, plus the possible variations in the location of this canal,[4] present the surgeon with numerous anatomical combinations. Guild[5] called attention to gaps in the osseous facial canal. The granulation packed cells adjacent to the fallopion canal may become attached to the nerve sheath and may simulate VII nerve tumors. Surgical trauma to the nerve may result during the exploration. About the mastoid antrum, the usual surgical landmarks may be entirely obscured when the granulations completely fill it. Dislocation of the ossicles, especially the incus, may occur during the removal of this fibrous tissue. For this reason an operating loupe is routinely used when examining the mastoid antrum and the retrofacial region and during the removal of granulations in these areas.

The present regime of treatment of temporal bone infections has been antimicrobial therapy, early myringotomy when secretions are present or suspected in the middle ear, adenoidectomy in infants, or an adenotonsillectomy in older patients, a window operation of the maxillary sinuses when a suppurative process is present which has not responded to medical treatment, and a simple or cortical complete mastoidectomy if necessary. Bacterial studies are made on all cases with aural discharge and the sensitivity to therapeutic agents determined in order that the most suitable drug may be used in the systemic treatment. The white blood count and differential are routine. Each case is followed by repeated audiometric examinations, age and men-

tality permitting. Stereo x-rays of the mastoids are taken when the aural discharge does not cease in a few days or there are clinical recurrences. It is considered that the nasopharynx, the nasal accessory sinuses, the eustachian tube, the middle ear, and the mastoid and apical cells are all involved in some degree in these infections.

Postoperative treatment, except for Case 1, has been similar. Since 1954 the following local treatment has been used in all cases requiring a cortical mastoidectomy. After the complete surgical exenteration of all mastoid cells, including the anterior and posterior zygomatic cells, and the involved retrolabyrinthine ones which may extend medially well into the petrous portion of the temporal bone, a No. 10 catheter is sutured into the postauricular incision, and brought out through the mastoid dressing. This catheter is used to introduce 2 cc of a suspension of terramycin 250 mgs, chloromycetin 250 mgs and polymyxin 50 mgs in 60 cc of normal saline with the pH 6. This treatment is given every three hours day and night for nine days. In addition, antibiotics are administered orally for five or six days.

In selected cases irradiation to the nasopharynx is used, but only after a thorough adenoidectomy has been surgically performed, removing all the lymphoid elements possible from the fossae of Rosenmuller under direct vision.

REPORT OF CASES

CASE 1. Patient was a 24 year old white male soldier admitted to Tokyo Army Hospital November 13, 1953. He stated his left ear began discharging five months previously. He denied prior ear discharge. Clinical examination revealed the left external canal to be filled with pus. The tympanic membrane was thickened. A small indefinite perforation was present below the umbo, midway to the annulus. A culture of the aural discharge revealed Proteus mirabilis, sensitive only to chloromycetin. Mastoid x-rays showed diffuse haziness of the mastoid cells with poor definition of the septa. These findings were deemed compatible with mastoiditis. On the sixth hospital day a swollen and tender area was noted over the left mastoid. Also the left angle of the mouth drooped a little and the left cheek crease was lessened. No palpable weakness of the left facial nerve was present. The left aural discharge ceased. While under observation, it was noted the patient could not taste with the anterior two-thirds of the tongue, and the left corner of the mouth continued to sag. There was an average loss of 28 decibels in the speech range in the left ear.

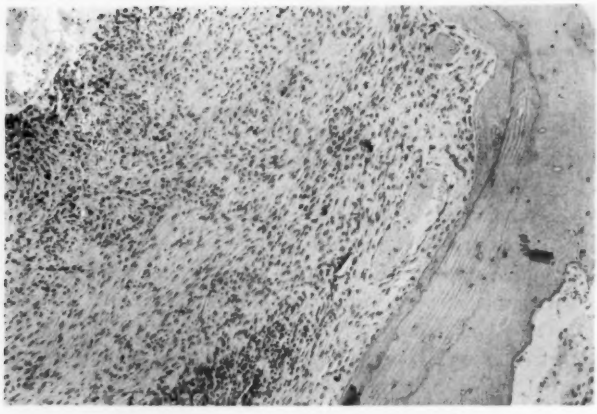

Fig. 1.—(Case 2) Photomicrograph of tissue from right mastoid showing mature fibrous tissue with an infiltrate of chronic and a lesser number of acute types of inflammatory cells. Vascular channels are prominent. x 200.

On November 25, 1953, a simple or cortical mastoidectomy was performed. No gross purulent secretion was present. The pneumatization of the left mastoid was quite extensive. The perifacial cells were carefully removed. No stimulation of the facial nerve occurred. The lining of the mastoid cells was definitely thickened. The tip cells contained a couple of cyst-like structures. The postauricular incision was closed with a rubber dam drain.

The pathological examination of the mastoid cells shows cancellous bone fragments whose medullary spaces show areas of lymphocytic infiltration. The tip cells show connective tissue infiltrated with mononuclear cells and lymphocytes. The diagnosis was chronic mastoiditis.

The postoperative course was interesting in that within two days there was noticeable improvement of the facial paresis in that the patient was able to whistle. In sixteen days the ear was dry, the incision healed, and the tympanic membrane showed a small perforation as noted preoperatively. Following a local tonsillectomy, the perforation closed and the average hearing loss 35 days postoperative was ten decibels in the speech range.

Comment: This patient presented several clinical symptoms which in the pre-antibiotic era would have urged immediate mastoid

exploration. However, with the reliance being placed on medical therapy in 1953, there was considerable reluctance to perform surgery.

CASE 2. This was a 36 year old white female with a two months' history during which time there were six exacerbations of ear pain and temperature, with increasing deafness in the right ear. Each episode had been treated with antibiotics. The following clinical picture was present February 17, 1956: The patient stated that the right ear felt full. The tympanic membrane was very thickened but intact. A myringotomy was performed and the middle ear aspirated. No fluid was present. The middle ear space seemed completely filled with swollen, thickened membrane. Inflation of the right eustachian tube was not successful. Sagging of the posterosuperior canal wall was present. There was an average loss of 33 decibels for air conduction in the speech range. Stereo x-rays revealed haziness throughout the well-pneumatized right mastoid with disruption of the bony trabeculae There was an absence of pain, temperature and leukocytosis. No response to the caloric test was obtained in the right ear. Differential diagnosis between chronic infection and tumor could not be made. The remainder of the history and physical were noncontributory, except there was a significant history of allergic symptoms and numerous positive skin tests had been obtained several months previously. The allergist thought the eustachian tube blockage could be accounted for on an allergic basis. Allergic controls and treatment were instituted several months before mastoid surgery was contemplated.

On February 24, 1956, a right complete cortical mastoidectomy was performed. The postauricular approach was used. As the mastoid cortex was removed, organized material bulged through the opening. Frozen section of this tissue was reported to be inflammatory material. The cellular development extended posterior to the lateral sinus. Many of the bony septa were markedly softened and could be removed by curettage. There was an accumulation of pus in the tip cells and in an area overlying the lateral sinus. The mastoid antrum was completely and solidly blocked with hyperplastic tissue. The incus was not viewed nor was the facial nerve uncovered; however, there was twitching of the right face when working about the mastoid tip. The right middle ear was inspected and seemed to be filled grossly with the same type of material as filled the mastoid cells.

Bacterial studies of the secretions from the tip cells showed Staphylococcus albus sensitive to several antibiotics, but insensitive to penicillin. Fungus cultures were negative after 24 days.

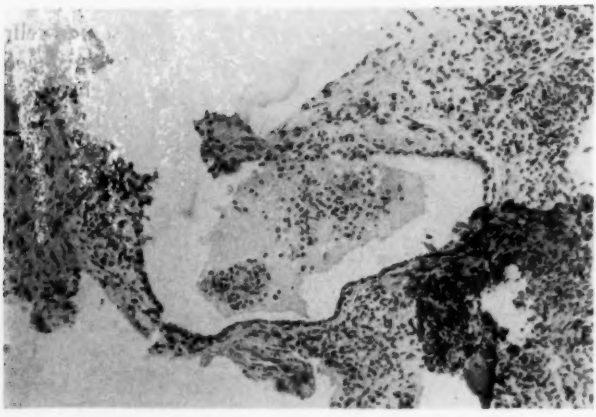

Fig. 2.—(Case 2) Photomicrograph of tissue from right mastoid showing fibrous tissue beneath the epithelial lining of a mastoid cell. The epithelium is seldom identifiable in the specimen. x 200.

Microscopic examination of the fragments of granulation tissue showed both recently formed and fairly mature fibrous tissue, an infiltrate of chronic and a lesser number of acute types of inflammatory cells and numerous vascular channels. Several fragments of bone exhibited fraying of the bony margins and an increase in osteoplastic activity presenting a picture of early osteomyelitis. The epithelial lining of the mastoid cells was seldom identifiable. Evidence of specific inflammation was not present. Fungus stains were negative (Figs. 1 and 2).

Postoperative therapy was used as outlined above. There was complete cessation of ear symptoms. One month after surgery the hearing in the right ear showed an average decibel loss of seven decibels in the speech range and a 30 decibel loss for the 4,000 and 8,000 frequencies. Five months later examination of the right tympanic membrane showed much less thickening, the landmarks were plainly visible, the eustachian tube was open, and the hearing was above the 10 decibel level for all frequencies.

Comment: From a clinical and audiometric standpoint the edematous process in the mucosa of the middle ear was reversible.

Case 3. Patient was a four and one-half year old white female who developed measles on February 5, 1956. Two weeks later a left

acute peritonsillar abscess developed, which was incised and drained on February 21. Child was on terramycin therapy. Seven days later aural discharge (beta streptococcus on culture) and some swelling and fluctuation over the left mastoid area was noted. White blood count was 21,200 with 81 per cent neutrophils. X-rays of the left mastoid showed the cellular development to be retarded with some sclerosis present. The cell walls were thickened and the over-all mastoid had a hazy appearance. The findings were consistent with the diagnosis of acute left mastoiditis.

On February 28, 1956, a left cortical mastoidectomy was performed. A small subperiosteal abscess was present over an area of eroded mastoid cortex. Many small cells were encountered which were packed with granulations. Between these were layers of very eburnated bone. The mastoid antrum was completely filled with thickened membrane and granulations. No gross suppuration was present in the mastoid cells. There was no secretion in the middle ear. A No. 10 catheter was sutured to the periosteum. The postoperative regime was employed as above described. Examination of the nasopharynx revealed a large mass of adenoid tissue.

Microscopical examination of the tissue fragments from the left mastoid showed granulation tissue composed of recently formed fibroblasts and new blood vessels. The tissue was infiltrated with lymphocytes, mononuclears, and fewer numbers of neutrophiles. The bony fragments exhibited frayed margins and an increase in osteoclastic activity in some areas. No areas of specific type inflammation were observed (Fig. 3).

Cultures from the left mastoid showed Beta hemolytic streptococcus sensitive to antibiotics except dihydrostreptomycin. Previous cultures from the throat and left ear showed a similar organism.

On April 27, 1956, patient had an adenotonsillectomy. Subsequent audiograms showed an average loss of five decibels in the left ear. The right ear maintained normal hearing throughout this infection.

Case 4. This seven year old white female developed an acute exacerbation of a chronic right ear infection December 21, 1955. The discharge continued until the latter part of February 1956, at which time there was a conductive hearing loss, a thickened right tympanic membrane, and x-ray studies were interpreted to show a destructive process in the right mastoid.

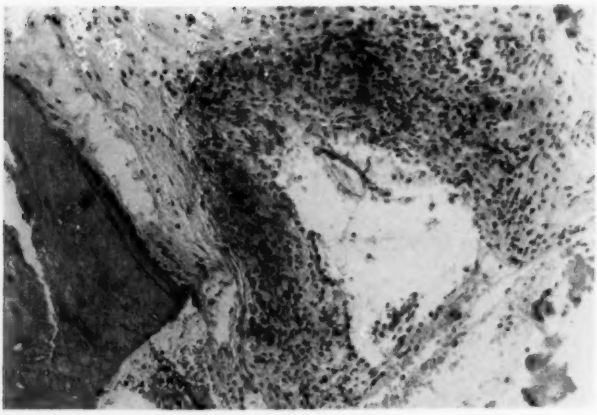

Fig. 3.—(Case 3) Photomicrograph of tissue from the left mastoid showing granulation tissue composed of recently formed fibroblasts and new blood vessels. The tissue is infiltrated with lymphocytes, mononuclears and fewer numbers of neutrophiles. x 200.

The past history was a lengthy one of many episodes of earache, temperature, and varying periods of inflammation and discharge since the child was three years of age. Some fifteen episodes had been recorded and treated by various antibiotics. Patient had an adenotonsillectomy two years previously at five years of age.

On March 13, 1956, a right cortical mastoidectomy was performed, using the postauricular approach. The mastoid cortex was removed with a cutting motor-driven burr. A large cavity was encountered just posterior to the mastoid antrum, which extended nearly to the sinodural angle. This corresponded to the area on the x-ray that was interpreted to be one of bony destruction. About this cavity the cellular development consisted of many small cells. These were filled with thickened lining membrane and granulations. As the deeper retrofacial cells were removed, a formed structure was encountered lying in close apposition to the bony lateral sinus wall. The bone about this structure was removed. This mass of tissue had a smooth outer capsule and was attached along its anterior side to the mid-third of the vertical portion of the facial nerve. The facial nerve was identified by a nerve stimulator. The course of the vertical portion of the facial nerve curved approximately six millimeters posterior to the usual expected course. Facial contractures occurred

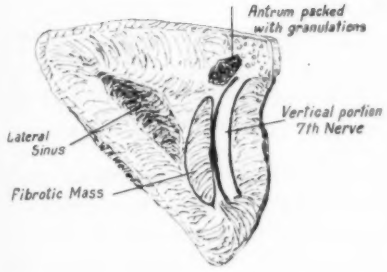

Fig. 4. — (Case 4) Sketch shows decompressed vertical portion of right VII cranial nerve with the fibrous mass attached to the posterior aspect of the nerve. The granuloma developed in the retrofacial cells. The nerve curved posteriorly approximately 6 mm from the usual vertical position. As viewed through 2½ power operating loupe.

when the stimulator was applied below the point of attachment. However, no reaction occurred when the stimulator was applied above the point of attachment. This was thought probably due to trauma to the nerve during its exposure and definite identification of the nerve structure (Fig. 4).

Frozen sections of the most posterior side of the bean-shaped mass indicated it to be fibrous tissue. The mass was then teased from the vertical portion of the nerve. Inferiorly the nerve was uncovered as far as the stylomastoid foramen and above to the horizontal portion. The mastoidectomy was completed. The incus and the incudomalleolar joint were viewed. The posterior zygomatic cells were packed with granulations. These involved cells were adjacent to small granulations on the posterosuperior quadrant of the tympanic membrane. A myringotomy was done and the middle ear space was clear of secretions. Gelfoam was placed over the decompressed facial nerve and a No. 10 catheter sutured into the wound. The patient awoke from the anesthetic with a complete paresis of the right facial nerve.

Pathological study of the tissue from the posterior zygomatic and mastoid tip cells showed tissue fragments to be composed largely of granulation tissue formed by a proliferation of fibroblasts and small vascular channels. An infiltrate of chronic inflammatory cells had occurred in this tissue. Numerous irregular spicules of bone were present which presented an intact generalized architecture, but in several areas exhibited fraying of the margin of the bone, presenting a picture of mild chronic osteomyelitis.

The mass taken from the posterior surface of the facial nerve was composed of mature fibrous tissue containing numerous vascular channels and a mild chronic inflammatory cell infiltrate. No neural elements were present.

The postoperative examination in May 1957, fifteen months after the cortical mastoidectomy, showed the right tympanic membrane to be intact, the average decibel loss in the speech range to be 12.5 decibels, in the high frequencies to be 17.5 decibels, and the right facial paralysis had recovered with hardly perceptible residual.

CASE 5. Patient was a ten year old white male who was admitted to this hospital on March 14, 1956, with a severe, acute, suppurative, bilateral otitis media and mastoiditis. Pain had been present for two days. Purulent discharge was present in both external auditory canals. The tympanic membranes were inflamed, thickened, and all landmarks were lost. The hearing was 2/20 spoken voice bilaterally. There was considerable tenderness over the lower half of the left mastoid area. Patient was acutely ill, with a temperature of 103 F., white blood count 21,150, 81 per cent neutrophils and 19 per cent lymphocytes. Hemoglobin was 13.05 grams. Stereo x-rays of the mastoids showed haziness on both sides. A radiolucent area was present near the left tip. Cultures of the discharge from each ear showed Proteus vulgaris and Pseudomonas aeruginosa.

The past history indicated patient had had measles at fifteen months of age, and later had scarlet fever, chicken pox and mumps. In 1953 patient received six radium treatments to the nasopharynx because of bilateral conductive deafness. There had been periods of bilateral aural discharge. X-rays taken at that time compared similarly to the ones on the present admission.

Medical treatment improved the local and general condition. The aural discharge was reduced and the hearing improved to the 25 to 30 decibel level. The radiological picture again showed haziness of both mastoids and on the right there appeared to be some breaking down of the cells.

On March 29, 1956, fifteen days after admission, a bilateral cortical mastoidectomy was performed. The postauricular approach was used. The right side was operated first, as the x-ray studies seemed to indicate this to be the more severely involved side. As the mastoid cells were uncapped they were found to be packed with granulation tissue. The lateral sinus lay in an anterior-posterior direction. The

postsinal, tip, retrofacial and retrolabyrinthine cells were extensively pneumatized. These cells were filled with granulations. The bone between the mastoid cells was very hard. Suction in the retrofacial area and on the anterior side of the mastoid tip caused movement of the right side of the face. The posterior zygomatic cells and the mastoid antrum were packed with granulation tissue. The incus was not seen. Throughout the right mastoid were many small yellow cysts. A No. 10 catheter was sutured to the periosteum and skin. The cavity was partially filled with Neocorten® ointment and the remainder of the incision closed with vertical mattress 3-0 black silk sutures.

The patient was repositioned and the left ear prepared and draped. The postauricular approach was used. On this side the posterior portion of the lateral sinus was quite superficial and the sinus lay in an anterior-posterior direction. There were large cells below and posterior to the lateral sinus, in continuity with the tip cells. The radiolucent area on the x-ray was a single large cell packed with granulations. In the retrofacial area a large mass, measuring approximately 16 mm in length, 6 mm in width, and 3 mm in depth, was encountered. This mass lay directly posterior to the vertical portion of the facial nerve. Stimulation of the upper portion of this mass caused facial contraction. The bony walls over the lateral and posterior sides of this structure were carefully removed. A biopsy of the posterior inferior portion of this mass was taken, and a frozen section was reported as showing fibrous tissue. No nerve cells were noted. This fibrous mass was intimately associated with the vertical portion of the facial nerve. Further removal was not attempted. The mastoid antrum and posterior zygomatic cells were solidly packed with firm granulations. In the tegmen area the contents of one of the cells was in direct contact with the dura, no intervening bone being present. A No. 10 catheter was sutured into place, the cavity partially filled with Neomycin® and cortisone ointment, and closed. The patient's general condition remained good during this rather lengthy procedure. Upon reacting from the anesthetic, the facial movement was normal on each side (Fig. 5).

The pathological specimen was composed largely of granulation tissue formed by a proliferation of fibroblasts and small capillaries, and was infiltrated by a variable number of all types of inflammatory cells. Spicules of bone exhibited some fraying and an increase in osteoclastic activity at the bony margins. Cultures of the mastoid operative sites showed no growth after four days.

On May 4, 1956, a re-adenotonsillectomy was performed. The right ear was still moist and the left tympanic membrane appeared

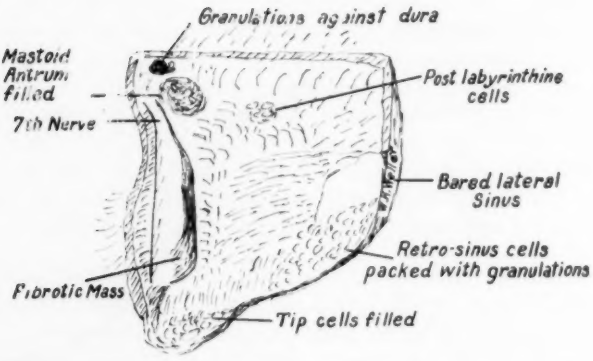

Fig. 5.—(Case 5) Sketch shows decompressed left facial nerve with fibrotic mass on posterior aspect. Viewed by 2½ power operating loupe.

healed. The audiogram showed an average loss of ten decibels in each ear.

The follow-up clinical examination on March 15, 1957, nearly one year later, showed scarring of both tympanic membranes. Both ears were dry. The audiogram showed 0 decibel loss in the left ear and 12 decibel loss in the right ear in the conversational range.

CASE 6. Patient was an eight year old white male admitted to the hospital because of severe deafness. The parents stated the deafness had been noted three years previously. There was no history of discharge from either ear but he had had earaches on three occasions. A previous adenotonsillectomy had been performed. Examination on admission revealed a child who was obviously very hard of hearing. Tonsil tags were present bilaterally. There was a large amount of postnasal discharge. Both tympanic membranes were intact but thickened. The right one showed a funnel-shaped healed perforation located centrally, which appeared to be adherent to the medial wall of the middle ear. Audiograms showed a severe mixed type hearing loss averaging in the speech range 48 decibels on the right and 63 decibels on the left. X-ray examination of the sinus and chest was normal. Mastoid x-rays showed moderate cellular development with uniform haziness throughout.

On March 8, 1957, patient had a re-adenoidectomy. Bilateral myringotomies were done and thick mucus aspirated from each mid-

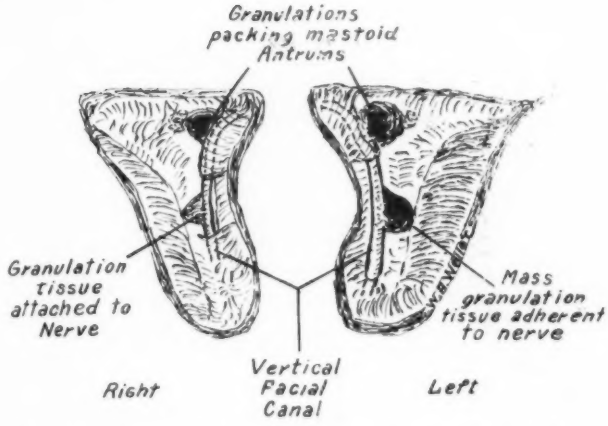

Fig. 6.—(Case 6) Sketch of each mastoid cavity showing decompression of facial nerves. The masses of granulation tissue were attached to the posterior aspect of each nerve near the middle of the vertical portion. The fibrous tissue on the left side was much firmer than that on the right. As viewed through 2½ power operating loupe.

dle ear. The eustachian tubes were inflated. The hearing improved following this surgery for two to three weeks and then began to decrease. Repeat x-ray studies showed no clearing of the mastoid cells.

On April 8, 1957, bilateral cortical mastoidectomies were performed. The left side was operated first. The postauricular approach was used. The mastoid cells were packed with granulations. The mucosa of the epitympanic and middle ear space was quite thickened. As the retrofacial cells were removed, a soft tissue mass was uncovered. This lay at the level of the facial nerve and was in direct continuity with the vertical portion of the nerve at about its mid area for approximately 7 mm. The course of the facial nerve, below the horizontal portion, gradually turned laterally and further in its course the fallopian canal turned posteriorly. Most of the tumor mass was removed from the posterior aspect of the nerve and the neurilemma slit above and below the mass for approximately 5 mm. The decompressed nerve was covered with gelfoam, a No. 10 catheter sutured in place, and the incision closed.

The right mastoid was explored. Numerous cells were filled with granulations. While removing the retrofacial cells, a bony cavity was encountered in about the same relative position as that containing the

TABLE I

(CASE 6) AUDIOGRAMS

DATE	AVERAGE DB. LOSS		AFTER POLITZERIZATION	
	RT.	LT.	RT.	LT.
6 March 1957	48	63		
7 March 1957	45	65		
8 March 1957	Re-T & A, Myringotomy and Aspiration of Mucus			
19 March 1957	30	36		
22 March 1957	32	30		
26 March 1957	18	20	13	18
29 March 1957	15	15		
3 April 1957	15	25	13	15
5 April 1957	15	18		
6 April 1957	20	35		
8 April 1957	Bilateral Cortical Mastoidectomy			
19 April 1957	33	43		
22 April 1957			20	36
26 April 1957			15	32
27 April 1957	Irradiation to Nasopharynx Total 300 r air to each side			
(Summer Camp)				
30 July 1957	43	35		

Further observation is indicated.

tumor mass on the left side. The bone was removed about this mass and stimulation showed the mass to be in direct continuity with the facial nerve. The nerve was decompressed 2 to 3 mm above and below. The mass was then removed from the posterior aspect of the nerve with minimum manipulation. The usual postoperative regime was followed (Fig. 6).

Postoperative examination showed no disturbance of function of either facial nerve. While the frozen section of the tissue removed from the left facial nerve was reported as showing nerve cells, the fixed slides showed collagenous connective tissue containing some chronic inflammatory cells. No nerve tissue was visualized.

The tissue removed from the mastoid cells on either side consisted of fragments of bony tissue showing sclerosis and surrounded by rather loose fibrous connective tissue containing moderate numbers of chronic inflammatory cells, including plasma cells and eosinophils. One area shows cholesterol clefts.

Patient received 300 r irradiation to each side of the nasopharynx from April 27, 1957, to May 2, 1957. Table I shows the results of the

audiometric examinations. Both tympanic membranes are intact, but further observation and treatment is indicated and will undoubtedly improve the hearing.

CASE 7. This eight year old white child was admitted May 31, 1957, with a left facial paresis of 16 days' duration. There is an indefinite but positive history of left ear infection manifested by recurring bouts of pain, occasionally depressed hearing, and at times some elevation of temperature over the past several years. The left ear has never discharged. X-ray examination shows marked sclerosis in the periantral region with haziness of the other mastoid cells. The hearing by audiometric test is normal. The left tympanic membrane is thickened but intact.

On June 1, 1957, a left cortical mastoidectomy was performed under endotracheal anesthesia. The postauricular approach was used. The periantral cells were quite sclerotic. The lateral sinus was deeply placed and many cells lay posterior to the sinus. The pneumatization of the left mastoid was very extensive. The cells varied greatly in size, many of them being very small, interspersed with others much larger. Many of these cells were completely packed with granulations. The post-sinus cells seemed to connect with those in the mastoid tip and the other numerous perifacial cells. The cells lying about the vertical portion of the facial canal were examined carefully for bony gaps. All of the cells adjacent to the nerve seemed to be separated by a thin layer of bone. Several of these cells were filled with granulation tissue while others escaped involvement. The vertical portion of the facial nerve was decompressed. Stimulation of the horizontal portion of the nerve with the nerve stimulator produced facial movement. The posterior zygomatic cells were small and numerous, many packed with granulations. These were exenterated, exposing the incudomalleolar joint. A myringotomy was performed and the middle ear found free of secretions. The exposed portion of the facial nerve was covered with gelfoam. A No. 10 catheter was sutured in the incision and the usual postoperative regime followed (Fig. 7).

The incision healed without infection. The movement of the left face began to return about seven days postoperatively and has gradually improved. The hearing four weeks after decompression of the left facial nerve was at a ten decibel level.

Pathological examination of the tissue removed from the left mastoid showed some degree of resorption of the bony fragments and these were associated with fragments of fibrous connective tissue con-

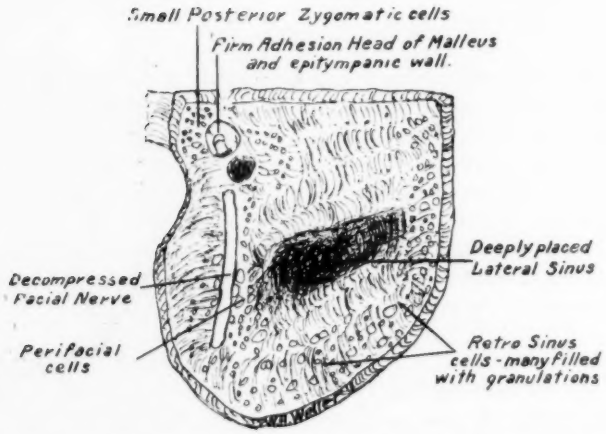
Small Posterior Zygomatic cells
Firm Adhesion Head of Malleus and epitympanic wall.
Deeply placed Lateral Sinus
Decompressed Facial Nerve
Perifacial cells
Retro Sinus cells - many filled with granulations

Fig. 7.—(Case 7) Sketch of decompressed left facial nerve. The perifacial cells were small in size but very numerous. No bony gaps were found in the vertical portion of the facial canal. The mastoid was extensively pneumatized with the lateral sinus deeply placed. As viewed with 2½ power operating loupe. Patient had preoperative left VII nerve paresis for 16 days.

taining moderate numbers of lymphocytes and plasma cells. There was no evidence of acute inflammation.

Comment: Kettel[6] suggests that preoperative VII nerve palsy occurring in association with chronic otitis media always indicates that a radical mastoidectomy be performed. In the light of the experiences with Cases 1 and 7, the author recommends that the vertical portion of the facial nerve be decompressed in the cavity of a cortical mastoidectomy through the postauricular approach. This preserves the middle ear structures but still permits the removal of areas of osteitis in the mastoid cells about the nerve.

Case 8. This 25 year old white soldier was admitted January 24, 1957, with a history of pain behind the left ear of ten months' duration. The left ear felt stopped up. There was slight tenderness over the left mastoid region. Patient stated, to his knowledge, the ear had never discharged. Previous therapy had consisted of an 11 day course of penicillin four months prior to the current admission and a ten day course just preceding this admission. The hearing was normal. X-rays showed diffuse clouding of an extensively pneumatized left mastoid.

On February 4, 1956, a complete cortical mastoidectomy was performed, using the postauricular approach. As the mastoid cortex was removed, the contents of the cells bulged under pressure. All cells were filled with extremely firm granulations. As the mastoid antrum was approached, the fibrosis became extremely dense. This stopped about the middle of the dome of the horizontal semicircular canal. The mastoid antrum was lined with moderately thickened mucosa. No discharge was present. The left middle ear was free of fluid. Routine postoperative therapy was employed. The hearing in both ears remained normal.

Pathological study of the tissue removed from the mastoid cells and near the antrum showed fibrous connective tissue and rather dense accumulations of chronic inflammatory cells, primarily lymphocytes and plasma cells, with occasional neutrophilic leukocyte. No definite epithelial lining was observed. The bony spicules showed marked irregularity along their surface. Tissue cultures showed no bacterial growth after seven days.

CASE 9. This patient was a 13 month old white child admitted April 15, 1957, with a history of an intermittent discharge from the left ear since early February of this year. Initial cultures showed B. Streptococcus. Therapy consisted of penicillin, streptomycin, terramycin, and finally ten days of chloromycetin. Later cultures showed a Staphylococcus aureus which was sensitive to all tested medications. X-ray studies indicated an absence of air in either mastoid. There was no history of discharge from the right ear.

On admission the white blood count was 26,850. The temperature was normal. X-ray examinations of the mastoids showed a punched-out area on either side, indicating an absence of air in the mastoid cells. The child's general physical condition appeared excellent despite the father's statement that there had been a ten pound weight loss during the past two months, associated with periods of skin rashes and diarrhea.

On April 16, 1957, a bilateral cortical mastoidectomy was performed. The left side was explored first, using the postauricular approach. The mastoid cells and antrum were solidly packed with fibrotic granulations. Examination of the left tympanic membrane showed a 1 mm perforation of the pars tensa at five o'clock. A No. 10 catheter was sutured into the incision. The right ear was prepared and draped. The right drum was intact but thickened. After a myringotomy, thick tenacious mucus was aspirated from the middle ear space.

Exploration of the right mastoid showed a similar pathological process as had been encountered on the left side. The extensive pneumatization of both mastoids was about equal. The postoperative regime as above outlined was followed by an uneventful course.

Pathologically the tissue from each mastoid showed rather loose edematous fibrous connective tissue with large numbers of plasma cells and eosinophils associated with histiocytes. Cultures from the right mastoid showed no growth in three days; from the left mastoid, Staphylococcus aureus.

CASE 10. This five year old white female was admitted with a small red spot just behind the postauricular crease of the left ear. This had been present for three days. The temperature was normal. White blood count was 8,000. The left tympanic membrane was intact and no aural discharge was present. X-rays of the left mastoid were hazy. There appeared to be an area of bone destruction in the posterior portion of the mastoid area. There was a history of previous ear infections which seemed to have been controlled with antibiotic therapy. The preoperative audiogram showed an average loss of 13 decibels in the speech range.

A left cortical mastoidectomy was performed June 7, 1957, employing the postauricular approach. The area of redness and swelling showed no suppuration. The pneumatization of the mastoid cells was very extensive. Many of the cells were packed with granulations. Posterior to the mastoid area, an area of the skull the size of a dime was eroded and filled with dense granulations attached to the dura of the medial cranial fossa. The bony plate over the upper half of the left lateral sinus had been entirely eroded, and dense granulations were adherent to the dura of the lateral sinus. The necrotic bone was removed until the bone bled readily. The lining membrane of the mastoid antrum was quite thickened. The facial nerve was not exposed; however, stimulation of the nerve occurred as the retrofacial cells were removed. The exposed dura was covered with gelfoam and a No. 10 catheter sutured into the periosteum and skin. The usual postoperative regime was followed.

Pathological examination of the material overlying the lateral sinus and dura of the medial cranial fossa showed islands of acute inflammatory cells embedded in fibrin and blood clot associated with granulation tissue composed of fibroblastic cells, small blood vessels and acute and chronic inflammatory cell infiltrate. Sections of the mastoid cells show bony trabeculae being actively resorbed by osteo-

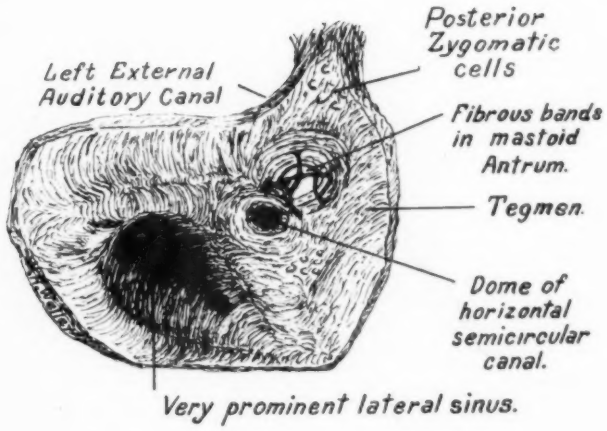

Fig. 8.—(Case 11) Sketch shows fibrous bands crossing mastoid antrum. The large prominent superficial lateral sinus simulated by x-ray an area of bony destruction. As viewed through 2½ power operating loupe.

clasts associated with a rather mature fibrous tissue matrix infiltrated by lymphocytes and plasma cells.

Cultures from the left mastoid showed a Staphylococcus albus resistant to penicillin and terramycin.

On July 10, 1957, an adenotonsillectomy was performed. An audiogram on July 30, 1957, showed an average loss of 11 decibels in the left ear. The left tympanic membrane was healed and intact.

Case 11. The patient was a 42 year old white male, retired Air Force officer, with a long history of left ear difficulty. While flying in 1943 he had pain and a stopped-up feeling in the ear. The ear was lanced and inflated several times. In 1945 he received radium therapy while in England and was retired from the service in that year because of otitis media and asthma. He received periodic inflations until 1956. Recent allergic studies showed a great many sensitivities to pollen and foods. In August 1956 he developed periods of vertigo and "blacked out," requiring hospitalization. The left ear was considered to be the cause of these symptoms. Examination showed a healed perforation of the left tympanic membrane. In early January 1957 the color of the tympanic membrane became a very dark blue. Stereo x-rays of the mastoids showed a peculiar punched-out area just anterior

TABLE II

BACTERIOLOGY OF CASES PRESENTED

CASE NUMBER	PREOPERATIVE STUDY	OPERATIVE STUDY
1	Proteus mirabilis	No growth
2	Staph albus	Staph albus. Fungus, culture and stains negative
3	Beta hemolytic Strep	Beta hemolytic Strep
4	Beta hemolytic Strep, aerobacter aerogenes, B. coli, Staph aureus and Pseudomonas aeruginosa	No culture reported
5	Proteus vulgaris, Pseudomonas aeruginosa	No growth after 4 days
6	No aural discharge	
7	No aural discharge	
8	No aural discharge	No growth after 7 days
9	Staph aureus	Right: No growth after 3 days Left: Staph aureus
10	No aural discharge	Staph albus
11	No aural discharge	No growth in 3 days

to the left lateral sinus. There was a moderately severe conductive deafness which improved by inflation as much as 40 decibels. Benefit lasted varying periods of time up to 24 hours and then the hearing dropped to the pre-inflation level. An exploratory cortical mastoidectomy was advised and performed on February 1, 1957. The postauricular approach was used. A few cells extending posterior from the mastoid antrum toward the sinodural angle were filled with granulations. The lateral sinus was quite superficial. The cortical bone overlying the lateral sinus was intact and appeared normal. The mucosa of the mastoid antrum was quite thickened and the cavity was bridged by strands of fibrous tissue (Fig. 8). As these were removed, the incus was exposed. The middle ear and antrum contained thick tenacious mucus. A No. 10 catheter was sutured to the periosteum. The usual postoperative regime was followed.

Pathological examination of the mastoid tissue showed small fragments of bone surrounded by young fibroblastic tissue. Other fragments of loose vascular fibrous stroma contained large numbers of chronic inflammatory cells including many plasma cells and a few eosinophils.

Comment: The period of hemotympanum as indicated by the dark blue color of the tympanic membrane lasted two to three weeks.

TABLE III

MASTOIDECTOMIES

YEAR	HOSPITAL	CORTICAL	MODIFIED	RADICAL
		(Complete)	(Baron)	
1953	Tokyo Army Hospital	2	2	7
1954	Tokyo Army Hospital	5	2	13
1955	Tokyo Army Hospital	6	1	9
1955	Fitzsimons Army Hospital		1	
1956	Fitzsimons Army Hospital	23	1	18
1957	Fitzsimons Army Hospital (8 months)	22		18

With thick tenacious mucus being present in the middle ear and antrum at the time of the surgical exploration, it would seem that the type of materials in the middle ear varies from time to time. Certainly the stage is set for periods of hemorrhage to occur with loose vascular fibrous tissue in the mastoid cells. For this reason the author cannot agree with the idea that ideopathic hemotympanum is a distinct entity as indicated by Sheehy and McKibben,[7] but rather part of the picture which may occur with fibrosing mastoiditis.

Table II shows the results of the bacterial examinations of the above cases. The fungus studies of Case 2 were negative.

Table III shows the numbers and types of mastoidectomies performed since 1953. There has been a marked increase in the number of complete cortical mastoidectomies in 1956 and 1957.

SUMMARY

Fibrosing mastoiditis is the development of granulation tissue in the mastoid cells. This seems more pronounced with the administration of antibiotics in the treatment of acute, subacute and recurrent ear infections. In turn, the production of granulation tissue in the mastoid cells reduces the effectiveness of any type of medical treatment. The products of inflammation are often trapped, and drainage from the mastoid cells impeded. These products may be of a serous, purulent or hemorrhagic type or thick tenacious mucus. Most commonly the secretions, if found, are in the middle ear, but may be pocketed anywhere in the mastoid cells.

Usually there is an associated involvement of the middle ear and epitympanic space. The mucosa is edematous with an infiltrate of

acute or chronic inflammatory and connective tissue cells. This results eventually in varying degrees of adhesive otitis media and produces clinically a conductive type hearing loss. Reversing the inflammatory process in the lining membrane of the middle ear has a better chance of occurring when the surgical removal of the foci in the mastoid cells has been accomplished. The earlier this is done, the better the possibility of obtaining a nonsuppurative ear, an intact tympanic membrane, and of preserving the hearing (Cases 1, 2, 3, 4, 6, 9 and 11).

Some complications of fibrosing mastoiditis have been encountered. Granulomas attached to the middle third of the vertical portion of the facial nerve have occurred in three cases. These fibrous masses present operative risks, as the nerve itself is already bared of bone and may be injured from manipulations in the diagnosis and uncapping of the pseudo tumor (Cases 4, 5 and 6). Further, patients presenting with facial nerve paresis have recovered following a complete cortical mastoidectomy (Cases 1 and 7). Osteomyelitis of the adjacent portions of the skull have likewise shown favorable response following surgical intervention (Case 10).

Bacteriological studies have shown a variety of organisms to be present. Cultures of the tissue removed surgically from the mastoid cells have also shown no growth. Fungi or other specific types of inflammation have not been observed in the pathological specimens.

While the diagnosis of fibrosing mastoiditis is based upon the pathological examination of the removed tissue, its presence may be suspected with a history of an ear infection which has not responded to antibiotic therapy or other measures short of a complete cortical mastoidectomy, local evidence of inflammation of the tympanic membrane or haziness of the mastoid process by x-ray, combined with a conductive hearing loss.

The local postoperative treatment is designed to furnish as wide a spectrum as possible against the remaining bacterial infection.

It is evident from the above cited cases that the medical treatment by antibiotics needs the assistance of adequate and skillful surgery, at least until better therapeutic measures of caring for ear infections have been developed. The surgical emphasis can well be placed on the acute and subacute phase of ear infections, or acute exacerbations, before the ossicles and tympanic membranes have been destroyed or the fibrotic changes in the middle ear become irreversible.

FITZSIMONS ARMY HOSPITAL

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IX

PREVENTIVE DISSECTION OF THE NECK IN CANCER OF THE LARYNX

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When carcinoma attacks portions of the larynx other than the vocal cords metastasis to the regional cervical lymph nodes occurs frequently. It is in these lesions involving areas adjacent to the vocal cords that block dissection of the neck can be used to prevent loss of life from undetected lymphatic metastasis. Other factors, such as the degree of malignancy, the length of time it has existed and the possible resistance of the host to the malignancy, influence the spread of carcinoma to the lymphatics and lymph nodes, but the location of the laryngeal lesion should receive prime consideration. In lymphatic embolization cancer cells reach the regional nodes as their initial if not only destination, and while the lymph nodes provide fertile soil for growth they also serve as effective filters for the cancer cells. The presence of metastasis to lymph nodes is more closely related to the prognosis than is the size or duration of the primary lesion.

Rouviere[5] demonstrated that the lymph supply of the larynx was divided by the true cord into supraglottic and infraglottic collecting chains. The supraglottic chain forms a pedicle near the aryepiglottic fold and extends to the pyriform fossa, through the thyrohyoid membrane, along the course of the superior laryngeal vessels, and then divides into the internal jugular and subdigastric chain of nodes. The infraglottic group drain into the internal jugular chain by the following routes: 1) through the cricothyroid nodes on the cricothyroid membrane, 2) by way of the pretracheal nodes and 3) by drainage through the thyroid lobe.

Edema and induration of the neck, necrosis of cartilage, fixation of the laryngeal structures and ulceration increase the chances of lymphatic invasion and distant spread. In cancer of the larynx there can be little question that palpable cervical lymph nodes constitute a definite indication for neck dissection. It is also necessary to include elective dissection of the neck in certain instances when no nodes are

palpable particularly in lesions involving the epiglottis, subglottic areas, pyriform fossa, aryepiglottic fold, arytenoid and vallecula where laryngectomy combined with radical dissection of the neck has improved the over-all five year survival figures.

In a series of 236 cases of carcinoma of the larynx enlarged cervical lymph nodes were present in 174 cases (74 per cent) with this enlargement due to metastasis in 115 cases (66 per cent). In the other 62 cases with no palpable lymph nodes unilateral block dissection was done as an elective procedure either because of the extensiveness of the laryngeal lesion or its location in areas not confined to the vocal cords or ventricular bands and in 16 (26 per cent) of these cancer was histologically present in excised lymph nodes (Table I).

TABLE I

LARYNGECTOMY AND RADICAL NECK DISSECTION

	NUMBER OF CASES	METASTATIC CARCINOMA	
Palpable Nodes	174	115	(66%)
No Palpable Nodes	62	16	(26%)
Total	236	121	(55%)

The curability of cancer of the larynx parallels that of cancer in other parts of the body where the survival is highest when there is no metastasis, less with microscopic metastasis and least when palpable metastatic lymph nodes are present. Dissection of the neck performed at the time of laryngectomy has resulted in more five year survivals when the lymph nodes were not palpable than when it was performed in the presence of palpable lymph nodes or in two stages after the appearance of late metastasis to the lymph nodes. In laryngeal carcinoma palpable cervical nodes are generally due to metastatic disease, but this criterion is not entirely reliable for palpable nodes may contain no cancer and malignancy may exist in nonpalpable nodes. In this series needless dissection of the neck was performed in 8 per cent of the cases when no cancer was found in palpable lymph nodes, yet in contrast microscopic cancer was present in 26 per cent of the cases without enlarged lymph nodes. Surgical treatment is contraindicated when there is fixation of the cervical nodes and of doubtful value in the presence of bilateral firm cervical metastasis.

In this study the usual site of metastasis was the cervical lymph nodes on the side of the primary involvement, and the lymph node present on the cricothyroid membrane was rarely involved by cancer. Perhaps the fibrous cricothyroid membrane deterred this method of spread so that the lymph drainage from the interior of the larynx was not directed toward this node. One common factor in the majority of the cases with metastasis to lymph nodes was invasion of the laryngeal cartilages (cricoid, thyroid, arytenoid, base of epiglottis).

Unilateral block dissection of the cervical lymph nodes was performed either as a primary or secondary procedure and the presence of enlarged lymph nodes was considered evidence of metastasis and no biopsy was done prior to the definitive surgery. The principal reasons for withholding biopsy were the sense of false security that developed if an excised lymph node was reported as containing no carcinoma and the danger of implanting tumor.[2] While a single though representative lymph node might be free from cancer there might be metastasis in other nodes clinically insignificant, evident only microscopically and not detected by examination of a single node. Surgical scarring and interference with lymphatic drainage so that subsequent block dissection could not be performed as an inviolate block were other lesser factors in the decision against biopsy.

INCIDENCE OF CERVICAL LYMPH NODE INVOLVEMENT IN CARCINOMA OF LARYNX

In a series of 221 cases of carcinoma of the larynx in which 236 neck dissections were performed the incidence of metastatic nodal involvement was 56 per cent (Table II). This group included carcinoma in all portions of the larynx and structures contiguous with the larynx. It was not possible in all instances to determine from the laryngeal lesion at the time of operation the original site of the growth. In many the growth was quite large and involved practically the entire larynx and in assigning individual classification the lesion was not necessarily limited to the area designated, but rather invaded the area into which it was placed.

As anticipated, cancer extending beyond the confines of the interior of the larynx, i.e., the vallecula, arytenoid, aryepiglottic fold and pyriform fossa with or without invasion of the lateral pharyngeal wall had the greatest percentage of metastatic lymph cancer (60 per cent).

TABLE II

INCIDENCE OF CERVICAL METASTASIS IN RELATION TO SPECIFIC LARYNGEAL LOCATION

LOCATION	NUMBER OF CASES	NUMBER WITH CANCER IN NODES
Epiglottis	62	35 (56%)
Subglottic Extension	47	21 (45%)
Anterior Commissure	33	19 (58%)
Vocal Cord	29	16 (55%)
Pyriform Fossa	26	16 (62%)
Vocal Cord and Ventricular Band	14	9 (64%)
Arytenoid, Aryepiglottic Fold and Ventricular Band	8	6 (75%)
Vallecula	2	2 (100%)
Total	221	124 (56%)

In 62 cases of carcinoma of the epiglottis the cervical lymph nodes were the seat of the metastasis in 35 cases (56 per cent) with a five year survival rate of only 24 per cent in the latter group.

An unexpected finding was the high percentage (58 per cent) of cervical node cancer in those cases in which the lesion involved only the vocal cord and ventricular band and in others where the growth was limited to the vocal cord itself. These findings cast doubt on the assumption that carcinoma limited to the vocal cords or ventricular bands does not frequently metastasize. While it does not take into consideration the time element or other factors in the spread of carcinoma malignant lesions of the vocal cord cannot be dismissed as rarely metastasizing.

RELATION OF METASTATIC CERVICAL CARCINOMA TO PALPABLE LYMPH NODES

Of the total 236 neck dissections 174 were performed for palpable lymph nodes and 115 (66 per cent) were found to contain cancer. The size of the lymph nodes was not used as an indication for neck dissection as advocated by Reed[4] who considered only lymph nodes 2 cm or larger as metastatic. In this group dissection of the neck was performed for any palpable node regardless of its size. In many instances the nodes were small, shotty and freely movable. In other

TABLE III

CERVICAL METASTASIS DETECTED AFTER LARYNGECTOMY

LOCATION	NUMBER OF CASES
Vocal Cord and Ventricular Band	33
Epiglottis	23
Subglottic	13
Pyriform Fossa	4
Arytenoid	4
Extensive	1
Total	78 (33%)

instances there was fullness in the neck without definite palpable lymph nodes, while in others tenderness could be invoked over the anterior cervical chain or carotid sheath. When any of these findings were present neck dissection was performed. It was surprising to note that in some cases positive nodes were present, even though the lymph nodes were quite small and clinically appeared innocuous. The difference of several mm in size, one way or another, can hardly be used to provide an accurate basis for decision regarding dissection of the neck.

Another interesting feature was that the palpable lymph nodes sometimes did not contain carcinoma, whereas, a node that was smaller and not palpable, did contain cancer. When lymph nodes are enlarged even though they are inflammatory, dissection of the neck should be performed, for it is my feeling that when inflammatory cells have reached the lymph nodes there is likelihood that some of the carcinoma cells have also migrated along the inflamed and dilated lymph channels.

METASTATIC CERVICAL CARCINOMA WITH NO PALPABLE NODES

The late appearance of metastatic lesions in the absence of local recurrence indicates that cancer must have existed at the time of the original operation in impalpable lymph nodes. In this series 78 cases (33 per cent) developed metastatic cancer in the cervical lymph nodes after laryngectomy that had been performed when no nodes were palpable (Table III). These results are comparable to those reported

TABLE IV

LOCATION OF LARYNGEAL LESIONS WITH NO PALPABLE NODES YET NODES HISTOLOGICALLY CONTAINED CANCER

LOCATION	NUMBER WITH CANCER IN NODES
Epiglottis	5
Subglottic Extension	5
Pyriform Sinus	3
Vocal Cord and Ventricular Band	2
Arytenoid and Aryepiglottic Fold	1
Total	16 (26%)

by others.[1,3] In these cases the five year survival rate was only 37 per cent, demonstrating that the success of radical dissection of the neck is less likely when palpable metastatic lymph nodes are found after the primary surgery. In these 78 cases dissection of the neck performed at the time of laryngectomy would have removed the microscopic metastasis and enhanced the chances of five-year survival.

In view of the above figures, should one wait for the development of enlarged lymph nodes to perform block dissection?

In a group of 62 cases of cancer of the larynx in which there were no palpable nodes and laryngectomy was combined with dissection of the neck, 16 (26 per cent) were found to have histologic evidence of metastasis (Table IV). Serial section of the extirpated lymph node block is not routinely done, but would result in an even higher percentage of positive reports. While the follow-up in this group has not reached the five year mark preliminary observations indicate that the five year survival rate will probably be substantially increased for 12 out of the 16 are living, clinically free from disease two to six years.

Based on the cases with late detection of metastasis to lymph nodes and those with microscopic evidence of cancer in lymph nodes without clinical manifestations, 94 patients (40 per cent) with laryngeal carcinoma, had carcinoma in the lymph nodes with no clinical evidence of such spread at the time of the initial surgery, so that the question of whether neck dissection should be combined with laryngectomy in all patients that are satisfactory surgical risks should be considered.

The inclusion of dissection of the neck with laryngectomy does not increase the mortality and adds only slightly to the morbidity. Although it increases the time of the operation, this is a minor consideration in the over-all evaluation of the results. The time consumed means little as far as the risk to the patient is concerned and exerts its chief toll on the surgeon and his associates.

Since the risk to the patient of including unilateral dissection of the neck with laryngectomy is negligible, elimination of metastasis to lymph nodes in approximately 25 per cent of patients before it is clinically evident seems sound surgical judgment. The finding of microscopic evidence of cancer in lymph nodes that are not palpable confirms the clinical impression that malignant lesions which involve parts of the larynx other than the vocal cord and ventricular band require block dissection on the side of involvement. When the epiglottis or subglottic area was involved without palpable nodes, the percentage of metastatic nodes was approximately the same (25 per cent) and approached the percentage for the entire group as a whole.

Lesions involving the pyriform fossa yielded a 50 per cent incidence of metastatic carcinoma, while the lymph nodes in those lesions limited to the vocal cords and ventricular bands were microscopically positive in only 13 per cent. Probably some of these cases could have been salvaged later when the nodes became clinically enlarged, but the opportunity for survival was enhanced by performing the neck dissection at the time of the primary laryngectomy before there was clinical evidence of metastasis. Unilateral block dissection of the neck was performed in cases in which the carcinoma was extensive in the larynx or involved areas other than the vocal cords and ventricular bands.

BILATERAL NECK DISSECTION

In selecting the side of the block dissection when no lymph nodes were palpable, difficulty arose when the lesion was situated in the midline, such as the epiglottis and anterior commissure. Dissection of the neck was performed with laryngectomy on the side of the greater primary involvement and the opposite cervical lymph nodes were removed at a later date. When lymph nodes were palpable the initial dissection of the neck was performed on the involved side followed by block excision of the contralateral cervical lymph nodes later. It was considered unwise to include bilateral dissection of the neck with laryngectomy because of the hazard of increased morbidity and mortality incident to simultaneous ligation of both internal jugular veins.

In the 15 cases with simultaneous bilateral dissections of the neck there were no deaths, but the morbidity was increased.

Edema and cyanosis of the soft tissues about the face and neck developed, accompanied by severe headache and prolonged malaise. In some cases drainage of cerebrospinal fluid by lumbar puncture was necessary to relieve the increased pressure and stabilize the central nervous system. Removal of only one internal jugular vein when bilateral neck dissection is performed preserves the intracranial venous return, but allows lymphatics to remain and results in an inadequate operation. By allowing a lapse of time between the dissections of the neck the vascular system becomes adjusted to the unilateral deficiency in blood supply and can compensate so that the subsequent change in the vascularity from contralateral jugular vein removal is better tolerated, yet the completeness of the operation is not jeopardized. The time elapsing between the second neck dissection and the primary laryngectomy and block dissection varied. After the primary operative wound was healed, which in the average case required approximately three weeks, the second neck dissection was performed. In other instances the contralateral neck dissection was delayed from four to six weeks.

In some cases the cervical lymph nodes contained cancer bilaterally, while in others the carcinoma was present on the side of greater involvement and in still others the lymph nodes were involved on the side of lesser primary involvement. Bilateral dissection of the neck was performed in cases with long standing or extensive local disease, in those invading the midline in which lymph nodes were unilaterally enlarged and in those with bilateral palpable lymph nodes. In cases involving the midline bilateral neck dissection was done on the side opposite the palpable lymph nodes before the clinical development of enlarged lymph nodes to remove the chance of undetected carcinoma.

SUMMARY

In a series of 221 cases of carcinoma of the larynx in which 236 dissections of the neck were performed, the incidence of metastatic carcinoma in the cervical lymph nodes was 56 per cent. The highest percentage of regional lymph node metastasis occurred in those lesions which were extensive in the larynx, or those involving the arytenoid, aryepiglottic fold, pyriform fosssa or vallecula. In 62 cases of carciname of the epiglottis the cervical lymph nodes contained carcinoma in 56 per cent with a five year survival rate of 24 per cent.

In the 174 cases with palpable lymph nodes 66 per cent were due to metastatic carcinoma, establishing the fact that block dissection of the neck combined with laryngectomy, is indicated in the presence of palpable lymph nodes regardless of their size or clinical features.

Seventy-eight of the 236 cases (33 per cent) developed cervical metastasis after laryngectomy, when no lymph nodes were palpable at the time of the primary operation with a five year survival rate of 37 per cent.

Preventive dissection of the neck was performed in 62 cases with no palpable lymph nodes and in 16 (26 per cent) of these cancer was histologically present in the excised lymph nodes. Preventive dissection of the neck was elected in those cases in which the lesion in the larynx was extensive, had existed for a long time or involved areas other than the vocal cords and ventricular bands.

When the laryngeal lesion was situated in the midline, dissection of the neck was combined with laryngectomy on the side of greater primary involvement and the contralateral neck dissection was done as a second procedure after the primary healing which varied from three to six weeks following the original surgery.

Since approximately 25 per cent of patients with carcinoma of the larynx not confined to the vocal cords and ventricular bands have microscopic cancer in the lymph nodes with no clinical evidence of such spread and the figure becomes 40 per cent, when those cases with late evidence of metastasis are included preventive dissection of the neck combined with laryngectomy is indicated in these instances. The inclusion of unilateral dissection of the neck with laryngectomy does not materially increase the mortality or morbidity and the slight risk involved is out-weighed by the advantage to the patient.

1712 Locust St.

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X

DENTAL FACTORS IN MIGRAINE

REPORT OF A CASE

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AND

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Wolff,[1] Graham,[2] and others substantially agree on the symptoms of migraine. The headache is sometimes preceded by visual disturbances that last several minutes to a half hour. The patient may experience scotomata in any of the various visual fields. He may report combinations of the following: bright flashes; geometric designs; wavy vision; golden balls; stars; tesselations and serrations. His pupils may be dilated with transient impairment of vision, increased lacrimation, and photophobia. Other prodromal signs may include phonophobia, vertigo, nausea, vomiting and paresthesias of the face, hands and foot.

The headache is usually periodic, paroxysmal and unilateral. It is throbbing at first and may become a steady ache in the later stages. The pain lasts from a few minutes to several days and may be mild to severe in intensity. In a classical case, the patient is usually prostrate and avoids personal contacts. In 65 per cent of the cases,[3] the patient shows migraine ancestry. Classically, ergotamine compounds have a specific therapeutic effect.

REPORT OF A CASE

The following patient presented classic migraine symptoms:

In February, 1953, a 45 year old, tense, business executive complained of a severe throbbing pain over the left temporal area which appeared ocular in origin. Further injuiry revealed the classical migraine syndrome. He received symptomatic relief from ergotamine

compounds for fifteen years. His family history showed one other member with similar symptoms.

Slit lamp examination of the anterior segment of the eye was negative. Ophthalmoscopic examination showed no apparent pathology. Visual fields were of no value in the asymptomatic phases. The scotomata were only transitory in nature. Refraction showed moderate hyperopia with presbyopia.

Dental consultation revealed the true etiology of the migraine headaches. The patient had a dental malocclusion caused by ill-fitting partial upper and lower dentures. After a complete dental rehabilitation, the symptoms gradually diminished over a four month period. He returned at regular intervals for two years and was asymptomatic without the use of medication.

In August 1955, approximately two and one-half years later, the patient suffered an acute coronary episode. Treatment with vasodilators for the acute episode and subsequent daily administration of vasodilators for a coronary insufficiency have not precipitated any migraine attacks.

COMMENT

In a normal chewing cycle, one set of masticatory muscles contract isotonically while the contralateral or balancing masticatory muscles contract isometrically. The balancing muscles receive mechanical support from the teeth and condyle during mastication. When that support is missing, the balancing muscles (in order to stabilize the mandible) receive excessive stimuli through the neuromuscular reciprocal innervation via the central nervous system. These excessive stimuli exceed the normal physiological capacity of the balancing muscles and cause a compensatory partial or complete tetanic contraction.[4] The partial or complete tetanic contraction may lead to referred pain on the homolateral side of the head.[5]

SUMMARY

A case is reported, meeting the criteria of migraine headaches with ocular signs and symptoms. The patient discontinued medication and was relieved of symptoms after dental rehabilitation. The use of

vasodilators did not precipitate any additional migraine episodes. The pathology in the case was not similar to true migraine.

719 PARK AVENUE

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XI

THE EFFECTS OF HYPOXEMIA UPON THE ELECTRICAL RESPONSE OF THE COCHLEA

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Although a number of investigators have measured auditory sensitivity in human subjects under conditions of oxygen deprivation, the effects of reduced oxygen supplies upon hearing remain obscure. Data from several studies[3,5,10,11] indicate that the effect is deleterious, whereas data from several other studies[1,2,7-9] indicate that reduced oxygen has no effect upon hearing whatever. The resolution of this contradiction is not available through an examination of methodological differences among these studies. There were several instances where incompatible findings were reported in independent studies in spite of the fact that in each of these studies the same measure of auditory sensitivity and the same means for producing oxygen want were used. Apparently, inquiries concerned with the establishment of the relationship between oxygen tension in the lungs and hearing are inquiries prone to difficulty.

More consistent data have been obtained by a few investigators who turned to electrophysiological methods. In 1949 Wever, Lawrence, Hemphill, and Straut[12] reported an experiment on cats wherein the effects of oxygen want upon the electrical response of the cochlea were studied. By restricting the study to the cochlear response, these experimenters were able to obtain data about one of the necessary antecedents to hearing without concern for the complicated effects that oxygen deprivation certainly had on neural activity.

Wever et al reduced oxygen supplies by using a gas bag containing dilutions of air with nitrogen. Their preparations were maintained under deep anesthesia and were respired artificially. The results of this experiment indicated that the cochlea was highly resistant to oxygen want. Reductions from 21 (air) to less than 4 per cent oxygen

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(by volume) were required before any changes in the response were observed. Whenever a loss in the cochlear potential occurred, it appeared as a steady drop through time and amounted to as much as 40 db in certain instances when an animal was near death. Restoration of air produced only partial recovery. Although the stimulus employed was usually a 1000 cycle tone, other frequencies were studied. The effects indicated were found for all tones investigated.

In 1952 Wing, Harris, Stover and Brouillette[13] reported a similar experiment. These experimenters sought to refine their measure of oxygen supplied to the cochlea by measuring oxygen saturations in blood samples rather than by depending upon an analysis of inspired gas mixtures. With such a measure, errors arising in the specification of gas mixtures were eliminated. Furthermore, the measure was taken late in the process of oxygen carriage to the cochlea so that fluctuations in the depth and rate of respiration did not constitute additional sources of error.

The results of the study by Wing et al showed that the cochlear response was adversely affected by reductions in blood oxygen. Levels from 6 to 9 per cent were sufficient to affect the potential. These investigators concluded that the hair cells of the cochlea are not as resistant to hypoxia as had been believed. Their evidence also indicated that total recovery in the electrical response was possible in cats even though they had been subjected to 3 per cent oxygen for as long as 30 minutes. Partial recovery occurred in those preparations which had been subjected to levels below 3 per cent, but recovery seemed to be temporary.

Data from these two studies on the effects of reduced oxygen upon the cochlear response indicate that the response is adversely affected by reduced oxygen. Nevertheless, the variability of these data points out the need for further investigation. This is especially so in regard to the ability of the cochlear response to recover from the effects of hypoxia.[2] *

The purpose of the present experiment was to establish the minimum level of oxygen saturation in the arterial blood of the cat which would be sufficient to sustain the cochlear response without loss, and

* Oxygen levels reported by Wing et al. and Wever et al. are not comparable without conversion. The former reported levels in alveolar air, whereas the latter reported levels in ambient air. Accordingly, their findings are actually more divergent than the percentages reported here would indicate.

then to determine the lowest level of oxygen saturation which would still allow full recovery of the response once the normal level of saturation was restored. Because hypoxia was not induced beyond the physiological limit set by an animal's ability to sustain itself without assistance, such as heart stimulants and prolonged artificial respiration, an opportunity to observe compensatory behavior was afforded. Recovery from hypoxia was studied over extended periods in order to ascertain the nature of the damaging effects of oxygen lack upon the hair cells of the organ of Corti.

METHOD

Anesthetization. A solution of diallyl-barbituric acid and ethyl carbamate was injected intraperitoneally in a dosage of 1 cc per kilogram of body weight. Each cc of this solution contained the following: 0.1 gm diallyl-barbituric acid, 0.4 gm ethyl carbamate, 0.013 gm sodium hydroxide, and distilled water, q.s. Hydrogen-ion concentration was slightly alkaline (pH=8.0). Administration of the anesthetic preceded surgical operations by about 30 minutes, and thereafter produced a deep anesthesia enduring for as long as 60.

Surgical Procedures. A 5 cm mid-line skin incision on the ventral surface of the throat, posterior to the lingual artery, provided access to the trachea. The sternohyoid and sternothyroid muscles which normally cover it were cut by electro-thermal cautery or retracted by blunt dissection. A small transverse cut in the ciliated epithelium and connective tissue between two adjacent tracheal rings provided entrance for a tracheal cannula.

The tympanic bulla was approached laterally through an oval incision in the skin covering the parotid gland. This incision, extending about 5 cm, was in line with the coronoid process of the mandible and the base of the pinna. Division between two ligatures of the superficial temporal vein gave access to the parotid gland which was then elevated to expose its blood supply. This supply was ligated and the gland removed. Resection of the sternocleidomastoid, digastric, and stylohyoid muscles and removal of the periosteum exposed the bulla. Admittance by drilling made the round window membrane accessible for placement of a recording electrode.

Method of Stimulation and Recording. Stimuli were restricted to pure tones produced by the beating of two oscillators, one of which had a fixed frequency of 50,000 cps. The second was variable from

34,000 to 50,000 cps, thus giving the apparatus an effective range of frequencies from 0 to 16,000 cps. The signal from each of these oscillators went to a detector whose output was connected through a filter and an amplifier to a Daven attenuator (Type T-690 CR) with a range of 100 db, variable in steps of 1 db. Output from the attenuator fed into a power amplifier which drove the speaker (Western Electric 555 receiver). Aerial sound was conducted into the ear by a tube leading from the speaker. A cannula on the end of the tube was inserted into the external meatus and tied securely.

A closed bulla technique was employed. Two holes were drilled in the lateral surface of the bulla: One hole (diameter 2 mm) was located near the stylomastoid foramen, just ventral to it, to accommodate the wire from the active electrode on the round window membrane; and a second hole (diameter 5 mm) was located in an anterior position toward the jugular foramen to serve as an inspection hole during electrode placement. Through this hole the round window could be seen clearly. Both of these holes were fitted with rubber stoppers. Through the smaller stopper passed the recording electrode wire, and through the larger one passed a fine hollow tube which insured equilibration of air pressure within and without the bulla cavity, a precaution necessitated by the possibility of partial dysfunction of the eustachian tube. The closed bulla technique was utilized in order to eliminate the possibility of a reduction in the recorded response due to a short circuit between the active electrode and contiguous fluids which might accumulate from serum or condensation over extended periods.

The active electrode consisted of a piece of platinum foil which was soldered to the end of a nylon-coated copper wire. An inactive electrode made from a steel hypodermic needle was inserted in the superficial layer of the masseter muscle.

Cochlear potentials picked up from the round window membrane were amplified and then measured with a wave analyzer (General Radio, Type 736-A) operating as a selective voltmeter. Each experimental animal was isolated in an electrically shielded soundproof room. The diagram in Figure 1 includes the assemblage and arrangement of apparatus required to execute the methods of stimulation and recording indicated.

Oxygen Control. The amount of oxygen available through inspiration was controlled by having experimental animals breathe one

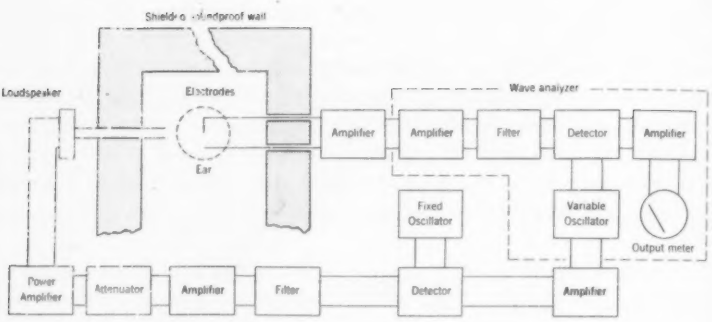

Fig. 1.—The assemblage and arrangement of apparatus required to execute the methods of stimulation and recording indicated in the text.

of four prepared gaseous mixtures maintained in separate storage cylinders. One cylinder contained ambient air, with oxygen content by volume equal to about 21 per cent, and the remaining three cylinders contained mixtures of oxygen and nitrogen, with oxygen at 10, 5, and 3 per cent, respectively. Although pressure in the storage cylinders varied from 1,500 to 100 psi, depending upon the amount of gas withdrawn, an oxygen-reducing valve on each cylinder held the pressure of withdrawn gases at 50 psi. Rubber tubes from each of these valves led to a common manifold which, in turn, led into a diluter demand oxygen regulator, activated by 50 psi. Valve settings on the manifold determined which one of the four storage cylinders supplied the demand regulator, and with the manifold, supply could be changed easily from one cylinder to another. The demand regulator was bled immediately following each change in supply so that the chambers of the manifold and the demand regulator contained the new mixture, absolutely.

A double Douglas valve (Krogh) situated between the demand regulator and the tracheal cannula provided a means for expired gases to escape the closed system with minimum contamination of inspired gases.

Positive and negative pressures required to activate the demand regulator and the double Douglas valve were measured and found to be 0.5 inch of water pressure. Considering the volume of the chamber of the Douglas valve, along with the rubber tubes leading into and out of it, and the normal rate and depth of respiration, it was calcu-

lated that an animal would breathe gas from a different supply cylinder within 12 seconds after changing to it at the manifold.

By opening a by-pass valve on the demand regulator, artificial respiration could be given simply by placing the hand over the exhaust side of the Douglas valve in a rhythmical sequence.

Blood Samples and Analysis. Samples of arterial blood were drawn from the carotid artery which was accessible through the incision made for tracheal cannulation. The artery was elevated from the surrounding tissue by blunt surgery; thereafter, all contiguous tissue was stripped from it with hemostats. To preserve normal action of the vagus nerve, it was separated carefully from the carotid. The exposed artery was cut obliquely midway between two ligatures 15 mm apart. The proximal aperture thus created allowed entrance for a flexible nylon catheter, 18 cm in length and 0.9 mm in diameter, which was inserted into the arterial orifice as far as the proximal ligature. After the catheter was tied in place the proximal ligature was removed for the duration of the experiment.

Each 3 cc blood sample was drawn through the catheter into a 5 cc syringe whose dead space contained an anticoagulant solution (heparin, 10 mg/ml). The syringe was attached to a hypodermic needle (No. 24) cemented within the free end of the catheter. A long catheter was used as a precaution against the danger of electrode movement during manipulations necessary to the withdrawal of blood. The barrel of each syringe was coated lightly with mineral oil, and as soon as the sample was drawn the syringe was capped. These two steps, oiling and capping, prevented contamination of the sample from oxygen in ambient air. Samples were chilled (40° F.) until determinations of oxygen saturations were made. Whenever samples were not being drawn during this experiment, a heparinized isotonic solution was forced slowly into the artery to prevent coagulation within the catheter and to restore blood volume.

A spectrophotometric method developed by Nahas[6] was used to analyze blood samples for oxygen saturations. So that analysis could be performed anaerobically, a special four chamber cuvette (Waters Conley) was used in conjunction with the spectrophotometer (Beckman, Model DU). The blood in each sample was analyzed within three hours of withdrawal, and each sample was hemolyzed just before analysis in order to obtain a clear solution for injection into the cuvette. Hemolysis was produced by adding to each blood sample a saturated

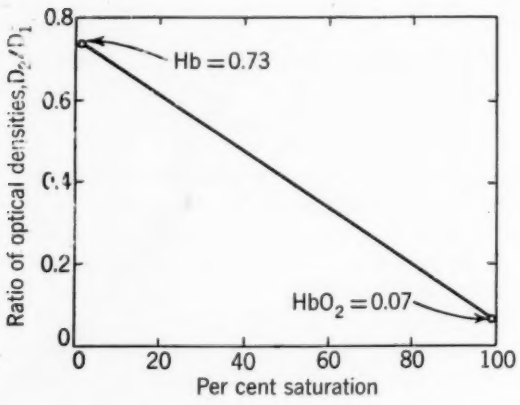

Fig. 2.—Calibration function for the optical densities D_2/D_1 for completely reduced and completely oxygenated arterial blood of the cat; where D_1 = optical density at 505 millimicrons, and D_2 = optical density at 605 millimicrons.

solution of saponin (Eastman-Kodak) in a quantity equal to about 1/100 of the volume of the sample. Thereafter, using a special rubber-tipped needle attached to the syringe containing the hemolyzed blood, three of the four chambers of the cuvette were filled. Density readings were obtained for wave lengths of 505 (D_1) and 605 (D_2) millimicrons. The empty chamber served in calibration procedures. The other three chambers gave readings at each of the two specified wave lengths. The mean value of density for each wave length was used in subsequent calculation. Between successive analyses the cuvette was flushed clean with distilled water.

Since the analysis of oxygen saturation depends upon the ratio of the optical densities D_1 and D_2 of reduced hemoglobin and oxyhemoglobin, these two ratios were determined first. From these ratios it was possible to calculate the oxygen saturation for any sample for which D_1 and D_2 were known. The first two samples withdrawn from each experimental animal were used to establish these calibration ratios. In the first sample hemoglobin was reduced by adding sodium hydrosulfite (10 mg/cc of blood). Oxyhemoglobin was produced in the second sample by placing it in a 50 cc flask containing 100 per cent oxygen for 15 minutes. Density readings were than taken at 505 and 605 millimicrons. The values of these two calibration ratios

(D_2/D_1 for the reduced sample and the fully oxygenated sample) define a function which describes the relationship between per cent oxygen saturation of hemoglobin and the ratio of optical densities. This function is presented in Figure 2. The significance of this function is that its slope represents the constant difference of the ratios of optical densities of completely reduced to completely oxygenated samples. Oxygen saturations in the samples withdrawn in this experiment were calculated from the following formula:

$$\% \text{ saturation} = \frac{X_{Hb} - X}{X_{Hb} - X_{HbO_2}} \times 100$$

where X_{Hb} = ratio of optical densities D_2/D_1 when the blood is totally reduced (this ratio = 0.73 in the present study); where X_{HbO_2} = ratio of optical densities D_2/D_1 when the blood is totally oxygenated (this ratio = 0.07 in the present study); and where X = ratio of optical densities D_2/D_1 of the unknown blood sample.

PROCEDURE

The anesthetized animal was brought into the shielded soundproof room in which the experiment was conducted. Here the animal was shaved with an electric clipper in those areas where an incision was to be made. So that the animal could breathe air freely during experimental preparations, the tracheal cannula was inserted first. Identification and elevation of the carotid artery followed, but catheterization was delayed. Temporal surgery led to the exposure of the round window. Afterward, the sound tube leading from the speaker was inserted into the external meatus and tied. Then the electrodes were put in place, and sound (1000 cps) was introduced to the ear at an intensity sufficient to produce a cochlear response of 50 microvolts. Finally, the nylon catheter was inserted into the carotid artery. During the two hours required to complete these preparations, all electronic equipment was turned on. On a dead animal used to test for the presence of microphonics no artifacts were obtained.

For most animals the cochlear response fell 2 or 3 db within 10 to 15 minutes after electrode placement and then stabilized. Only after stabilization were variations in oxygen availability begun. Three blood samples were withdrawn before deprivation. Two of these were used subsequently to establish the calibration ratios mentioned previously, and analysis of the remaining sample provided a measure of hemoglobin saturation after the animal had breathed air for several

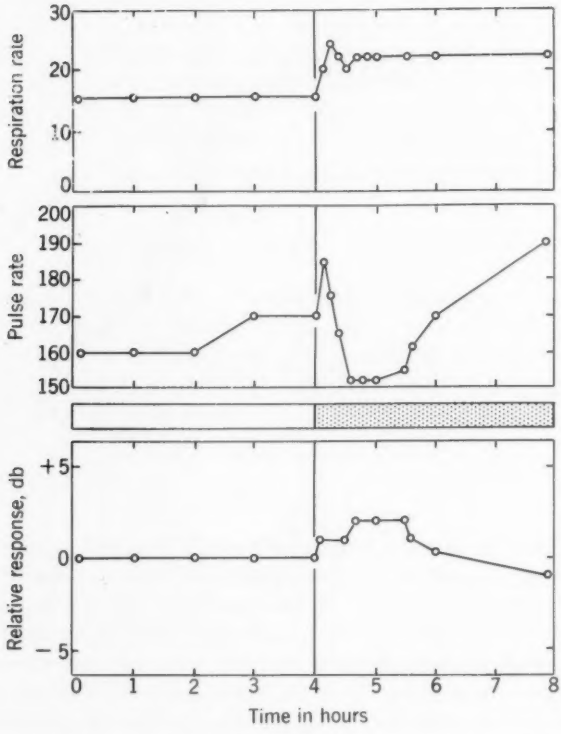

Fig. 3.—The response is given in db relative to the output prior to deprivation. Pulse is given in beats per minute; respiration in cycles (inspiration-expiration) per minute. The bar indicates oxygen availability: plain bar = air (O_2 about 21 per cent by volume); dots = O_2 at 10 per cent. Stimulus was a 1000 cycle tone.

hours. Additional samples were withdrawn during the temporal course of hypoxemia and recovery. Beside hemoglobin saturation and the cochlear response, data included records of changes in pulse and respiration.

Data were obtained early in this investigation which indicated that the cochlear response could be maintained without appreciable fluctuation ($\pm$3 db) for as long as 18 hours provided the animal did not suffer oxygen want. Removal of blood in a manner comparable to that employed on subsequent animals had no effect upon this

response. Therefore, in regard to animals in want of oxygen we may consider changes in the cochlear response to be the direct result of hypoxemia or of compensatory responses induced by it.

RESULTS

Data gathered on 12 cats in this experiment indicate that the cochlear potential is affected by hypoxemia. For the sake of simplicity, the results will be presented in three groups, one group for each of the three prepared mixtures of oxygen and nitrogen (O_2 at 10, 5, and 3% by volume).

When a gas mixture containing 10 per cent oxygen is breathed, the effect on the response is deleterious only after extended periods of time. Compensatory responses, especially those manifested in increased cardiac output and increased tidal volume, are adequate to sustain the response at its normal level for as long as two hours. Presented in Figure 3 are data on changes in the rates of pulse and respiration and changes in the response for a cat that breathed the 10 per cent mixture for four hours. This preparation was successful in compensating for decreased oxygen tension until the time of its death. Although pulse rate quickened initially, it fell to a level below its normal rate shortly after the animal began breathing the reduced mixture. Respiration rate also increased, but unlike the pulse, it remained high. The response showed a slight improvement (2 db) during the first two hours of deprivation and then fell off slowly until death. For this particular preparation, its reliance upon homeostatic mechanisms prevented any significant loss in the cochlear response.

The pattern just described is not unlike one observed for another cat that breathed the same reduced mixture for about two and one-half hours. Respiration rate doubled immediately upon inspiration of the reduced mixture, and, as before, the response showed an increase (2 db). Only in the last half hour of deprivation were the deleterious effects of oxygen want observed. The total loss in response amounted to 4 db, and if the pulse had not accelerated to a dangerously high rate, making the restoration of air advisable, a greater loss probably would have ensued. Nevertheless, upon breathing air the rate of respiration dropped sharply to the rate which prevailed prior to deprivation; and while the pulse rate declined slowly through the course of one hour the cochlear response recovered fully.

A third preparation subjected to the 10 per cent mixture behaved similarly, except that it was somewhat less successful in compensating

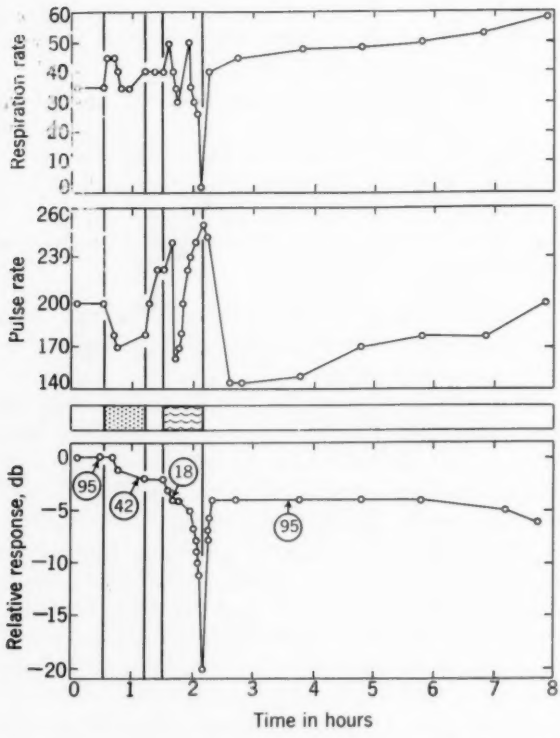

Fig. 4.—The response is given in db relative to the output prior to deprivation. Pulse is given in beats per minute; respiration in cycles (inspiration-expiration) per minute. The bar indicates oxygen availability: plain bar = air (O_2 about 21 per cent by volume); dots = O_2 at 10 per cent; waves = O_2 at 5 per cent. Stimulus was a 1000 cycle tone. Numbers within circles are Hb saturations.

for reduced oxygen tension. Respiration increased very little (about 20 per cent in rate), and except for a brief initial rise, the response fell off slowly through the two hour period of deprivation to a level about 4 db below the reference level. Restoration of air arrested the declining response but failed to produce any observable recovery in a one hour period.

The loss in output of the cochlear response for preparations that breathed the 5 per cent mixture was greater than the loss sustained by the group just described. In Figure 4 data are presented for a

preparation that was first subjected to the 10 per cent mixture and then the 5 per cent mixture. The length of deprivation for each of these mixtures was the same (40 minutes), but the effects observed were decidedly different. The loss was small (2 db) for the less severe level of deprivation, whereas during inspiration of the 5 per cent mixture the response fell to a level 20 db below the reference. The numbers presented within circles on the graph of the response indicate the hemoglobin saturation in per cent, as determined from blood samples. The rapid drop observed in the response indicates that hemoglobin whose saturation is only 18 per cent does not transport sufficient amounts of oxygen to the hair cells to allow them to maintain the cochlear potential unaffected; and this seems to be so in spite of the fact that the rates of pulse and respiration were considerably above normal during most of the period in which the response fell off.

Recovery was extremely rapid. Within five minutes after the restoration of air the pulse rate fell within the normal range (150-200 beats/minute) and the response showed a gain of 16 db, thus leaving a net loss of only 4 db. Notice, however, that further recovery was not observed at any time within the next five and one-half hours. During this long period ventilation was adequate to produce a level of hemoglobin saturation equal to that found prior to deprivation: furthermore, pulse rate was normal and the heart was strong. In view of these conditions it is especially significant that total recovery failed to occur.

A loss of 14 db occurred in the response of another preparation during a 30 minute period of deprivation on the 5 per cent mixture. The general pattern found for this animal resembled the one just described. Partial recovery occurred rapidly, but, as before, total recovery was not observed even though hemoglobin saturation was as high as it had been prior to deprivation. Hemoglobin saturation dropped from 95 to 22 per cent during deprivation and then increased to 92 per cent within ten minutes after the restoration of air. During the first hour after the end of deprivation the partially recovered response dropped 2 db. Thereafter, it remained steady at a level 8 db below the reference until death of the preparation five hours later.

On the third animal deprived of oxygen by breathing the 5 per cent mixture, three periods of deprivation were presented with recovery periods between them. Each deprivation period (two, two, and ten minutes, respectively) produced a greater loss in the cochlear potential than its predecessor. The losses were 2, 6, and 12 db, respectively. Total recovery was observed after the first and second losses,

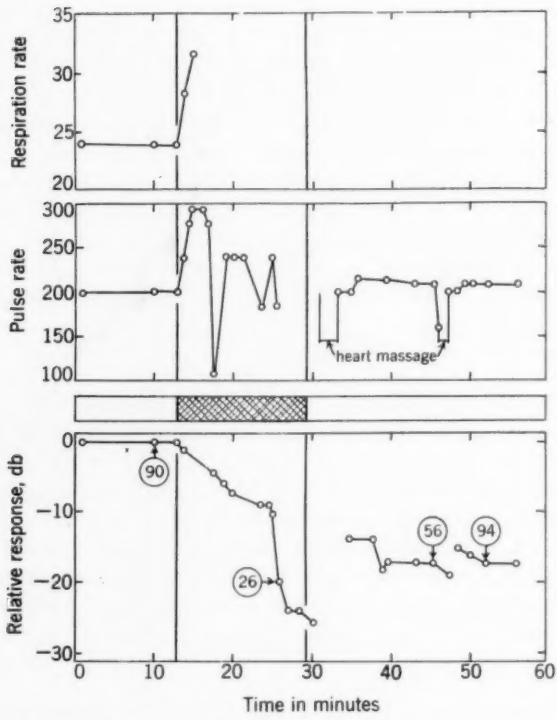

Fig. 5.—The response is given in db relative to the output prior to deprivation. Pulse is given in beats per minute; respiration in cycles (inspiration-expiration) per minute. The bar indicates oxygen availability: plain bar = air (O_2 about 21 per cent by volume); hatch = O_2 at 3 per cent. Stimulus was a 1000 cycle tone. Numbers within circles are Hb saturations.

but after the final loss a partial recovery which proved to be temporary was observed. What had been gained early in the final recovery period was lost three hours later. Observations on recovery show that twice the hair cells of this animal recovered fully from some state of want induced by a hemoglobin saturation of 18 - 22 per cent, a level of saturation which is the same as that found on two other preparations that failed to demonstrate full recovery. This problem will be discussed later.

Only limited observations were made on preparations subjected to the 3 per cent oxygen mixture because hypoxemia was so severe

that the animals could not maintain themselves without assistance. Maintenance of these preparations required artificial respiration and the administration of heart stimulants, such as adrenaline, or heart massage. Consequently, data gathered under these conditions are not comparable, strictly speaking, with those already discussed. Nevertheless, several interesting observations were recorded.

Figure 5 presents data collected from a preparation that breathed the 3 per cent mixture for the periods indicated. Because the changes in pulse, respiration, and cochlear response occurred very rapidly, the time axis on this graph is indicated in minutes rather than in hours. Unfortunately, efforts made to keep this animal alive interfered with the normal procedures used in noting the effects of oxygen want. Occasional gaps in data were unavoidable. When two successive points on this graph are not connected by a line, no observations were made during that interval.

As the graph of respiration shows, this animal stopped breathing within one minute after the onset of deprivation. All efforts made to restore natural breathing were unsuccessful, and so artificial respiration was administered until the time of death. The heart was unable to sustain its action without periodic massage. The cochlear response dropped about 10 db in the first 12 minutes of deprivation. Heart failure accounts for the precipitous drop thereafter. Hemoglobin saturation was about 26 per cent during the latter drop, but the absence of circulation makes the value of this determination questionable. The hair cells of the organ of Corti must have been in more serious want than they would have been if the heart had not stopped. The fact that hemoglobin saturation is affected by the extent of ventilation as well as by oxygen partial tension is exemplified in these data. With hyperventilation induced by artificial respiration, hemoglobin saturation did not go below 26 per cent, whereas levels as low as 18 per cent were recorded with the 5 per cent mixture on occasions when the animals were not respired artificially.

After the restoration of air, hemoglobin saturation increased from 56 to 94 per cent without effecting any increment in the cochlear response. Death prevented further observations.

The effects of compensatory responses on the cochlear response were demonstrated on another animal that breathed 3 per cent oxygen. Immediately after the 3 per cent mixture was introduced the response dropped abruptly (9 db). Following closely after this loss there was

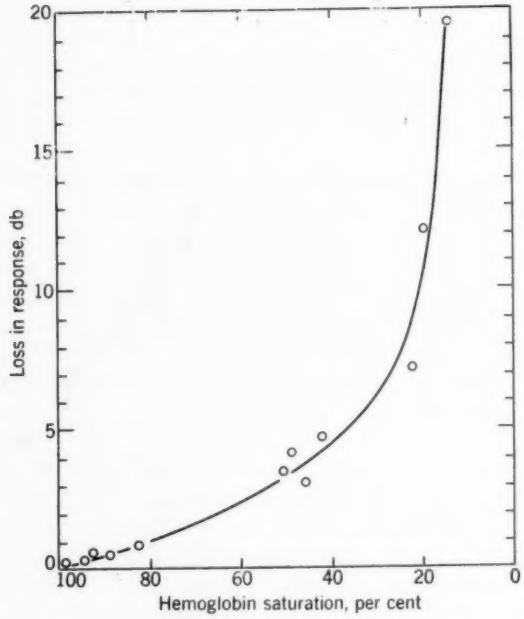

Fig. 6.—Maximum losses in the cochlear response during hypoxemia are plotted against hemoglobin saturation.

a gain of 7 db which we may attribute to an increase in the delivery of oxygen to the hair cells as a result of a quickened pulse induced by oxygen want and increased efficiency in oxygen exchange at the lungs as a result of artificial respiration.

During a second period of deprivation the response fell to a level 28 db below the reference even though the pulse was normal and respiration still continued artificially. After air had been inspired for 20 minutes the net loss in the output of the response was 7 db, thus giving a partial recovery of 21 db.

COMMENT

In spite of the differences which exist among cats in their susceptibility to hypoxia and their capacity to recover from it, the data

gathered in this experiment show that a definite relationship exists between the severity of oxygen want and the magnitude of the cochlear response. This relationship is represented by the function depicted in Figure 6. In this graph maximum losses occurring during hypoxia are plotted against hemoglobin saturation. Loss in the response is expressed in db relative to the cochlear potential output prior to hypoxia, and hemoglobin saturation is expressed in per cent, where 100 per cent represents maximum capacity for oxygen saturation (1 gm of Hb carrying 1.34 cc O_2 defines 100 per cent saturation). For those animals that sustained repeated intervals of deprivation only data gathered in the first of these spans are presented.

From Figure 6 it can be seen that the saturation of hemoglobin can drop to a rather low level before appreciable losses in the cochlear response become evident. A decrease in saturation from 90 to 80 per cent has little effect upon the output of the response. A reduction in saturation from 25 to 15 per cent, on the other hand, produces very marked effects. Based upon the nature of this function it looks as though the cochlear response is maintained without serious loss through a wide range of arterial oxygen saturation. It is only when hypoxia is severe that dire consequences are observed.

Although Wing et al made no effort to establish a functional relationship between arterial oxygen saturation and loss in the cochlear response, when their data are transformed into measures comparable to those employed in this study, it is clear that both sets of data are in essential agreement.

Oxygen is certainly among the more important substances necessary to metabolism in vertebrate cells. Failure in finding full recovery after periods of hypoxia suggests that oxygen is not only important to the generation of the cochlear response, but it also important to the life of these cells. Because full recovery is observed in the response following mild states of hypoxia we may assume that oxygen want under these conditions affects the process of generation of the potential without placing the hair cells themselves in serious jeopardy. Severe oxygen want, however, destroys some hair cells or produces irreversible changes in them and full recovery becomes impossible. By considering the relationship between the minimum level of hemoglobin saturation during hypoxia and the net loss in the cochlear response after normal amounts of oxygen have been restored, we can gain information about the extent to which hemoglobin saturation can fall without causing irreversible damage and loss.

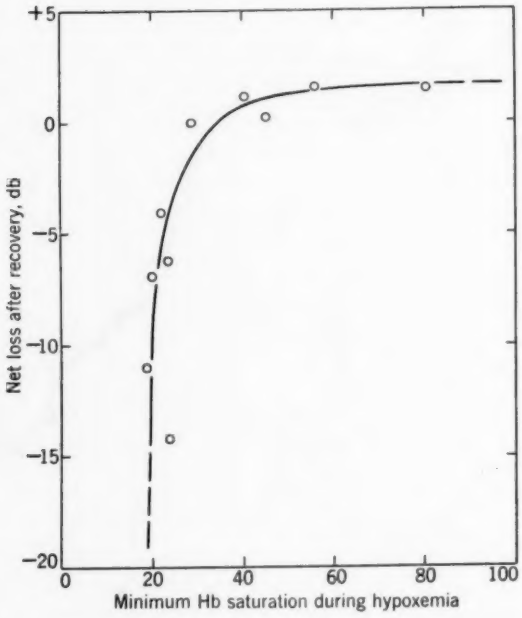

Fig. 7.—Minimum levels of hemoglobin saturation obtained during hypoxemia are plotted against the net loss in the cochlear response after the normal oxygen supply had been restored.

In Figure 7 the minimum level of hemoglobin saturation obtained during each span of hypoxia is plotted against the net loss in the cochlear response after normal oxygen had been restored. Hemoglobin saturation is expressed in per cent, where 100 per cent represents maximum oxygen saturation. Net loss in the response is expressed in db relative to the output of the response just before hypoxia was induced. A net loss of zero db, therefore, indicates full recovery. From this function it is apparent that whenever hemoglobin saturation falls below 35 per cent the response does not recover fully. After mild states of hypoxia (35 to 90 per cent), however, full recovery occurs. Points plotted above the zero db reference line simply indicate an enhancement in the output following the restoration of normal oxygen. Typically these enhancements were temporary. It should be pointed out that the net loss after recovery was determined by comparing the output of the response prior to hypoxia with the

maximum output obtained during the recovery period quite regardless of its duration. It should not be concluded from the curve presented in Figure 7 that a permanent gain of 2 db followed periods of deprivation wherein hemoglobin saturation fell to levels between 60 and 100 per cent saturation.

Since the cochlear potential recorded at the round window represents a type of summation of potentials generated by all of the cells acting in response to a particular stimulus, full recovery would be possible only if oxygen want failed to eliminate the contributions of any of these cells through cellular destruction. Partial recovery suggests that some of the hair cells suffered irreversible damage while others remained unaffected. In the event of partial recovery a situation prevails in which oxygen want reduces the cochlear response by interfering with the generation process and at the same time destroys some number of hair cells.

The hair cells do not have access to any appreciable amount of oxygen held in storage. Total oxygen reserves probably consist of what little the endolymph holds and perhaps some in adjacent cells. That this reserve supply is soon exhausted is clear. When the normal delivery of oxygen becomes impaired it is only a matter of seconds before the output of the potential declines. This does not imply, however, that these cells suffer from a constant level of hypoxia from the onset of deprivation throughout its temporal course. There are two reasons. First, time is required for the saturation of hemoglobin with oxygen to reach the minimum level set by the oxygen partial pressure in alveolar air. Limited observations made during this investigation suggest that about three minutes are required before hemoglobin saturation stabilizes. It is erroneous, therefore, to assume that a two minute period of deprivation on a 5 per cent mixture induces a level of hypoxia as severe as some other period of longer duration. For this reason it was not surprising to find that on two occasions the cochlear potential recorded from one of the preparations in this experiment recovered fully from two-minute periods of deprivation and not from a subsequent one which lasted ten minutes.

The second reason why the severity of hypoxia at the hair cells increases through the experimental period of deprivation is that 20 to 30 minutes are required before many of the tissues outside the ear are receiving the full effects of oxygen want. Extremely small amounts of oxygen are required by tissues of preparations during deep anesthesia and the metabolism of these cells is affected slowly. As the limited supply of oxygen which is associated with body fluids is depleted, the

oxygen tension differential between partially oxygenated capillary blood and contiguous tissues increases. The result of this increase is a greater loss of available oxygen to tissues other than the hair cells. Only after the whole preparation is truly hypoxic can we assume constancy of hypoxia at the hair cells.

These two temporal factors probably account for the characteristic drop in response. The abrupt loss observed immediately after the onset of deprivation reflects the first of these factors (stabilization of hemoglobin saturation), and the decelerated loss eventuating in stability reflects the second temporal factor (decreased oxygen to the hair cells as the needs of other tissues increase).

Wever et al did not report oxygen want in terms of arterial oxygen saturation. Consequently, direct comparison of their findings with those of the present investigation is impossible; but several observations deserve comment. Wever et al did not find full recovery in the cochlear response in any of their preparations. Recalling that their animals inspired dilutions of air containing from 4 to 0.5 per cent oxygen, failure of the response to recover is reasonable since data from the present experiment indicate that inspiration of a mixture containing 4 per cent oxygen (Hb saturation approximately 27 per cent) induces a state of hypoxia which is beyond the physiological limit which allows full recovery. The question of why mixtures containing between 10 and 4 per cent oxygen failed to affect the response in their experiment remains unanswered. However, preparations in their experiment were maintained under artificial respiration and therefore at a lower level of activity than those used in the present study, and, as a result, it is probable that smaller amounts of oxygen were required by tissues outside the ear, thus leaving proportionately more oxygen for the hair cells.

The present investigation, like those cited in its discussion, failed to control cardiac output, an important variable in studies of hypoxia. It is clear that if a constant volume of arterial blood circulated through the *stria vascularis* per unit of time then hemoglobin saturation would indicate fairly accurately the amount of oxygen supplied to the hair cells of the organ of Corti. In fact, however, volume is not constant. The primary determinant of volume per unit of time at the *stria vascularis* is cardiac output, and during hypoxia it is far from being constant. Cardiac output depends at least upon the pumping action of the heart, peripheral resistance, viscosity of the blood, blood volume, and elasticity of the arterial walls; and because these factors are difficult if not impossible to control, the usual procedure is to assume that

stroke volume remains constant. Essentially, cardiac output then becomes a simple linear function of pulse rate. In view of the fact that pulse rate is extremely unreliable as a measure of cardiac output, no effort was made to incorporate estimates of cardiac output in determinations of oxygen delivery.

The blood of cats differs from that of man in one important aspect. For equal volumes, hemoglobin concentration in cat blood is about 66 per cent of the concentration found in human blood. Accordingly, if all other parameters of importance which determine oxygen delivery to the hair cells were equated, then the cochlear potential generated in the human ear would probably be more resistant to oxygen lack induced by a particular mixture than the potential generated in the ear of the cat. Such an equation, however, ignores a host of complexities such as differences in effective areas of the lungs, differences in rates of respiration, differences in circulation rates, and differences in the proportion of blood volume to the number of hair cells. Accordingly, direct transfer of data obtained in this experiment to man is unjustified. Still, from the similarities evident in the process of oxygen carriage in man and cat, and an appreciation of the fact that hemoglobin saturation is by far the most critical factor in determining oxygen transport and delivery, there is little reason to think that the relationship existing between oxygen want and the cochlear response in the cat is widely different from the relationship in man: but more data are required before this statement ceases to be largely speculation.

SUMMARY AND CONCLUSION

Inquiry was made into the relationship between the level of hemoglobin saturation in arterial blood and the magnitude of the cochlear response for twelve cats that sustained one or more periods of hypoxemia induced by respiring one or more gaseous mixtures containing 10, 5, and 3 per cent oxygen, respectively. The cochlear response produced by a 1000 cycle tone was recorded continuously from the round window, and during the temporal course of hypoxemia and subsequent recovery, blood samples were withdrawn periodically from the carotid artery. These samples were analyzed later for oxygen saturation by means of a spectrophotometric technique (Nahas).

On the basis of this study the following conclusions are offered:

1. The cochlear response is resistant to oxygen want. A reduction in hemoglobin saturation from 100 per cent to 35 per cent reduces the magnitude of the response very little (within 5 db).

2. Levels of hemoglobin saturation below 35 per cent reduce the magnitude of the response drastically (up to 20 db).

3. Once normal oxygen is restored the cochlear response can usually recover its pre-deprivation output provided that hemoglobin saturation did not go below 35 per cent. For lower levels of saturation only partial recovery occurs.

4. In the event of partial recovery a stable output is achieved shortly after the restoration of normal oxygen, and this level is maintained without further recovery for extended periods (6 hours).

5. Partial recovery suggests that some number of hair cells suffered irreversible damage as a result of oxygen want.

6. The net loss in the cochlear response following oxygen deprivation is related to the severity of deprivation. The lower the level of hemoglobin saturation during deprivation the greater is the net loss observed in recovery periods.

7. On occasion, temporary enhancement of the response follows states of hypoxemia.

8. In instances of mild hypoxemia preparations can compensate for oxygen want with increased cardiac output and increased efficiency in oxygen exchange.

9. If the cat sustains a hearing loss during mild hypoxemia (100 - 35 per cent) it is not due to a markedly diminished cochlear response (maximum loss noted was only 5 db).

10. If the cat sustains a hearing loss after mild hypoxemia it is not due to the effects of oxygen want on the cochlear response since the small losses which may occur during mild hypoxemia are easily recovered after normal oxygen is restored.

11. If the cat suffers a hearing loss during severe hypoxemia (35 - 15 per cent) this loss is due in total or in part to a diminished cochlear response.

12. After severe hypoxemia if the cat suffers a hearing loss it is due in total or in part to the effects of oxygen want on the cochlear response since permanent (longer than six hours) impairment of the response persists.

13. Data obtained may not be directly transferred to man.

UNIVERSITY OF DELAWARE

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XII

THE EFFECTS OF AIR PRESSURE ON THE EAR

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Maximum auditory sensitivity depends partly upon a state of equal air pressure within and without the middle ear cavity. In the normal ear the eustachian tube provides a means for the air pressure in the middle ear to match continuously the air pressure in the external meatus, with the result that the tympanic membrane suffers neither deformation nor abnormal tension. However, when blockage of the eustachian tube occurs, as with congestion of the nasopharynx or infections of the middle ear, altered air pressure in the middle ear cavity can, and usually does, lead to a loss in sensitivity. In instances of marked departure from equality of pressures acting upon the tympanic membrane, severe and prolonged losses may follow. In the clinical situation such losses usually represent not only the effects of pressure changes, but also the combined effects of various extensions of infections. Consequently, the effects of pressure upon hearing must be studied on ears free of complicating disorders if a clear understanding of these effects is to be obtained.

Data from several studies[1-3] indicate that abnormal pressure on the ear of the cat, no matter whether it is introduced into the external meatus or the middle ear, has a deleterious effect upon the cochlear response. Moreover, positive and negative pressures produce similar losses. Typically, small positive or negative pressures ($\pm$ 4 inches of

From the Princeton Psychological laboratory. This research was aided by a contract (N6-onr?720-?) with the Office of Naval Research and by Higgins funds allotted to Prince on University. Permission is granted, for reproduction and use, by the United States Government.

water), introduced into either the external meatus or the middle ear cavity, decrease the magnitude of the cochlear response over the range of low and middle frequencies. The higher frequencies remain immune to the effects of pressure until relatively high deviations from the normal level occur (± 16 inches of water, and beyond).

Although positive or negative pressure acting upon the oval and round windows does affect sound transmission, its effect is small when compared with that which results from the same pressure acting upon the tympanic membrane. In each of the studies already cited the suggestion was made that altered air pressure in the external meatus or the middle ear cavity affected sound transmission mainly by changing the tension characteristics of the tympanic membrane. If this suggestion is correct, then, regardless of the magnitude of pressure changes, sound transmission should remain essentially unaffected as long as the tension of the drum membrane remains unaltered.

The experiment reported here was designed to test this hypothesis. To preserve normal tension of the tympanic membrane, pressure changes were introduced simultaneously into the external meatus and the middle ear. The cochlear response, used as an index of sound transmission, was recorded from each of 15 cats for each of several frequencies for both positive and negative pressures.

PROCEDURE

Each animal was anesthetized with a solution of diallylbarbituric acid and ethyl carbamate injected intraperitoneally in a dosage of 1 cc per kilogram of body weight. Tracheal cannulation was performed as soon as a surgical level of anesthesia was reached.

The tympanic bulla was approached laterally through an oval incision in the skin covering the parotid gland. This incision, extending about 5 cm, was in line with the coronoid process of the mandible and the base of the pinna. Division of the superficial temporal vein between two ligatures gave access to the parotid gland, which was then elevated to expose its blood supply. This supply was ligated and the gland removed. Resection of the sternocleidomastoid, digastric, and stylohyoid muscles and removal of the periosteum exposed the bulla.

Two holes were drilled in the lateral surface of the bulla. One hole (diameter 2 mm) was located near the stylomastoid foramen

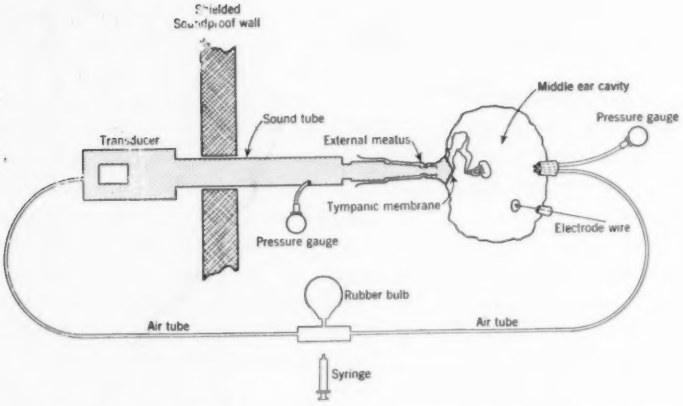

Fig. 1.—A schematic diagram of the apparatus used to produce equal positive or negative pressures in the external meatus and the middle ear.

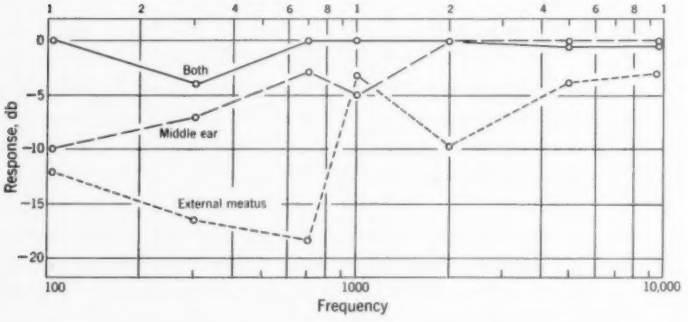

Fig. 2.—The effects upon the cochlear response of a positive pressure of 4 inches of water. Data on the middle ear from Wever, Bray, and Lawrence, 1942 (2), and on the external meatus from Rahm, Strother, and Crump, 1956 (1).

to accommodate the wire from the active electrode which terminated on a piece of platinum foil placed on the round window membrane. A second hole (diameter 5 mm), used initially for purposes of inspection, was located in an anterior position toward the jugular foramen and gave admittance to a pair of small tubes through which pressure changes were introduced and recorded in the middle ear cavity. Both holes were carefully stoppered. Through the smaller stopper passed the electrode wire and through the larger one passed the pair of small hollow air tubes of a bore of 0.034 inches. One of these small tubes introduced the pressure into the middle ear while the other one, attached to a pressure gauge, assisted in recording pressure changes. The arrangement is shown in Figure 1.

Tonal stimuli were generated by a General Radio Type 913-C beat frequency oscillator whose output was connected to a Daven Type T-693 attenuator. The output from the attenuator fed into an especially designed crystal transducer. A hard rubber tube which terminated in a cannula tied in the external meatus led aerial sound from the transducer to the tympanic membrane. This sound tube was pierced by a small air tube through which pressure changes could be made within the sound tube during aerial stimulation. Although with this arrangement the transducer was exposed to the changes in air pressure in the sound tube and external meatus, its design was such that these changes had no appreciable effect upon the output of sound. The air tube leading from the sound tube and one of the pair of tubes leading from the middle ear cavity were attached either to a common rubber bulb (for positive pressure) or to a common 50 cc syringe (for negative pressure). The small caliber of the pressure tube to the middle ear effectively attenuated sound from the meatal tube so that stimulation of the cochlea was accomplished by the conduction of sound through the normal route. A second pressure gauge was placed so as to provide a measure of the air pressure existing in the external meatus. Both pressure gauges were calibrated from —24 to +24 inches of water. The eustachian tube was clamped shut with a hemostat.

Cochlear potentials picked up from the round window membrane were amplified and measured with a General Radio Type 736-A wave analyzer operated as a selective voltmeter.

The intensity of each tone was adjusted initially to give a response of 50 microvolts at atmospheric pressure (zero). One of 10 pressures was then applied simultaneously to the external meatus and the middle

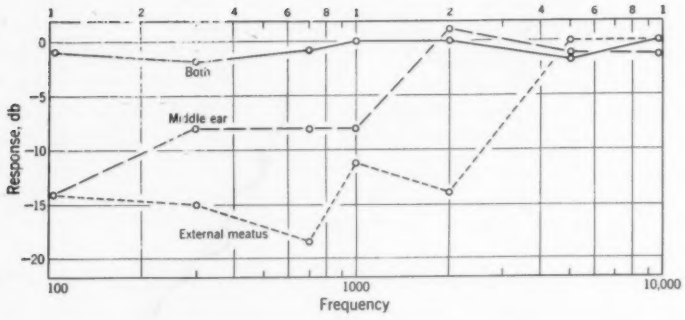

Fig. 3.—The effects upon the cochlear response of a negative pressure of 4 inches of water. Data on the middle ear from Wever, Lawrence, and Smith, 1948 (3), and on the external meatus from Rahm, Strother, and Crump, 1956 (1).

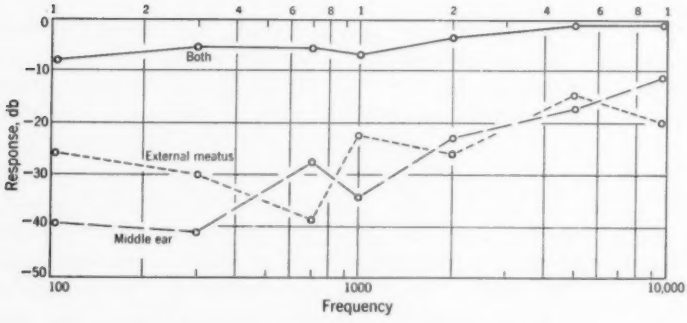

Fig. 4.—The effects upon the cochlear response of a positive pressure of 20 inches of water. Data on the middle ear from Wever, Bray, and Lawrence, 1942 (2), and on the external meatus from Rahm, Strother, and Crump, 1956 (1).

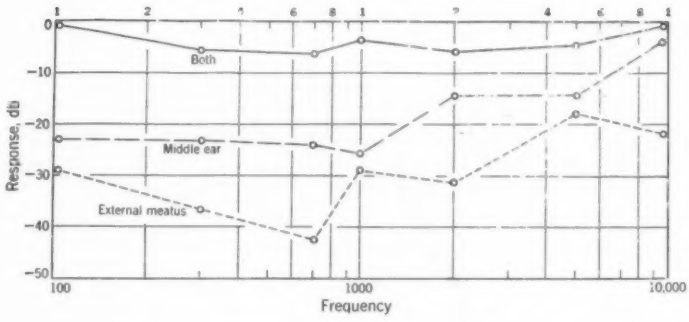

Fig. 5.—The effects upon the cochlear response of a negative pressure of 20 inches of water. Data on the middle ear from Wever, Lawrence, and Smith, 1948 (3), and on the external meatus from Rahm, Strother, and Crump, 1956 (1).

ear, and the magnitude of the cochlear response was noted. Seven frequencies were investigated. Both positive and negative pressures were used at 4, 8, 12, 16, and 20 inches of water. Periodic checks made at atmospheric pressure indicated that the magnitude of the cochlear response did not vary more than 2 db from the initial level (50 microvolts) through the course of the experiment. Animals were isolated in an electrically shielded soundproof room.

RESULTS

Data from representative cats are presented in Figures 2, 3, 4, and 5. In each of these figures the cochlear response is given in db relative to the output prior to the application of a pressure condition. For the sake of comparison, data on the effects of similar pressures applied only to the external meatus or only to the middle ear are included. These data were taken from the references cited. From Figure 2 it is apparent that when a positive pressure of four inches of water exists simultaneously in the external meatus and the middle ear, the cochlear response sustains no change, except within the limits of normal fluctuation. A positive pressure of 4 inches of water in the middle ear alone, however, does result in substantial losses in the response within the frequency range of 100 to 2000 cycles. Greater and more widespread effects occur when the pressure is applied in the external meatus.

Figure 3 presents data showing the effects of a negative pressure of four inches of water. The effects noted here are similar to those already discussed.

Data presented in Figure 4 show the effects upon the cochlear response of a positive pressure of 20 inches of water. Whereas no effect was observed for small positive pressures which were present simultaneously in the external meatus and the middle ear, losses up to 8 db were observed when the pressure was increased to 20 inches of water. Although the losses were fairly uniform and widespread, the frequencies beyond 5000 cycles remained immune to the effects of pressure. That the losses noted under this particular pressure condition were limited to 8 db probably means that the conductive mechanism of the ear was not severely hampered. Note the dramatic losses produced by the same positive pressure introduced solely to the external meatus or the middle ear.

Figure 5 presents data showing the effects of a negative pressure of 20 inches of water. Once again, the negative pressure produced losses similar to the ones produced by strong positive pressure.

COMMENT

The results given here represent about the average of the 15 animals studied. There were some animals in which the changes were greater, and others in which the application of pressures had no measurable effects. These differences are either true individual differences or, as we suspect, are due to a failure at times to keep the pressures on the two sides of the drum membrane exactly balanced. It is probable that even a slight imbalance, and one lasting only a short time, can produce changes in the mechanical properties of the membrane that will reduce its transmission in moderate degree.

In any event, the observed changes in transmission are small relative to the changes produced by large air pressures exerted on only one side of the membrane. The theoretical implication deriving from these data is that the differential pressure existing in the middle ear and the external meatus is the critical factor affecting sound transmission. The absolute magnitude of the pressures acting upon the tympanic membrane is of little consequence as long as they are matched and thereby relieve the membrane from physical distortion.

PRINCETON UNIVERSITY

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XIII

NEUROFIBROMA OF THE PARAPHARYNGEAL SPACE

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Although neurogenic tumors of the pharynx and neck are rare, the neurofibroma is more commonly seen than the meningioma or ganglioneuroma. It has also been designated as a perineural fibroma, perineural fibroblastoma, benign solitary tumor, and neurinoma, the latter designation being used by those who believe that the tumor arises in the cells of the sheath of Schwann, so that the term Schwannoma is used to designate its neurectodermal origin. However, most feel that it is a benign tumor of perineural connective tissue origin forming round or fusiform swellings on the course of larger nerves. It is important to understand that although the tumor is attached to the nerve sheath that nerve fibers do not pass through the tumor. Malignant degeneration rarely occurs, and may make itself known by preoperative paralysis. Microscopically the tumors are well encapsulated and composed of long slender cells, the long oval nuclei of which are arranged in palisades or whorls (Fig. 1). There may be central liquefaction necrosis and hemorrhage (Fig. 2). Multiple neurofibromata (Von Recklinghausen's disease) occurring in great numbers along small cutaneous nerves are related tumors, as are the acoustic nerve tumors.

Neurofibromas may make their appearance in the pharynx and have been designated as pharyngeal tumors by some authors. However, if they are covered by normal mucosa and outside the pharyngeal constrictor muscles and buccopharyngeal fascia (pharyngeal aponeurosis) they are more accurately described as being in the lateral or parapharyngeal space with the structures of the carotid sheath (Figs. 3, 4). These neurogenic tumors are characteristically slow growing and depending upon their size and location may cause dysphonia, a feeling of pressure, dysphagia, symptoms of the eustachian tube obstruction, and only in a few rare large tumors, respiratory obstruction or pain. Clinically the tumor must be differentiated from a chordoma, mixed salivary gland tumor, cold abscess, lipoma, and lymphoblastoma.

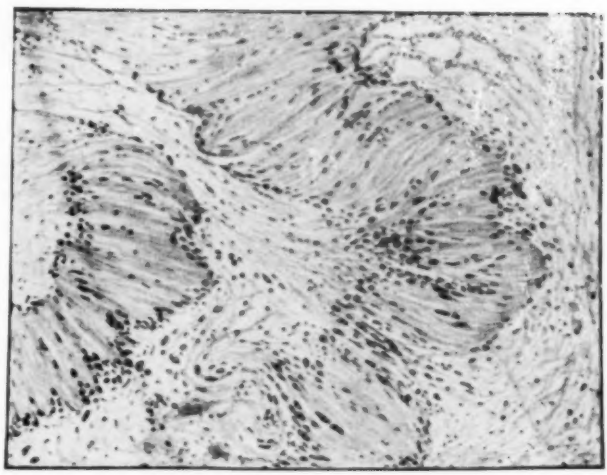

Fig. 1.—Neufibroma. Note arrangement of oval nuclei in whorls and palisades. (From Boyd, Textbook of Pathology, Lea and Febiger, with permission)

Fig. 2.—Neurofibroma showing central areas of liquefaction necrosis.

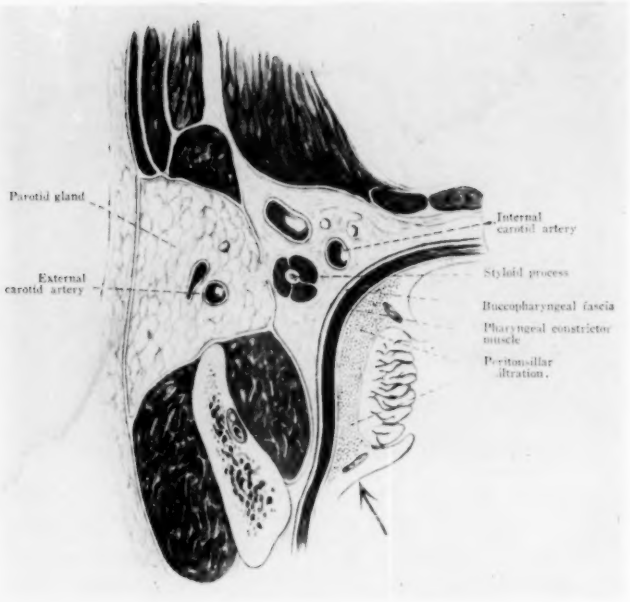

Fig. 3.—Partial cross section of head at tonsillar level to show parapharyngeal space. (From Kirschner, Operative Surgery, Lippincott, with permission)

The encapsulated benign neurofibromas are quite resistant to x-ray therapy and should be treated by total removal, either intraorally, or, if quite large, by an external cervical approach. Great care should be taken to avoid injury to the internal carotid artery or jugular vein or the glossopharyngeal, vagus, spinal accessory, hypoglossal, or cervical sympathetic nerves. Some authors have considered postoperative Horner's syndrome or other paralysis as unavoidable, thinking that fibers of the involved nerve pass through the tumor. However this seems doubtful. Some have stressed the need for preliminary ligation of the external carotid artery to minimize bleeding and preliminary tracheotomy to ensure an adequate airway. However, these additional surgical procedures are not invariably necessary as will be seen in the following case.

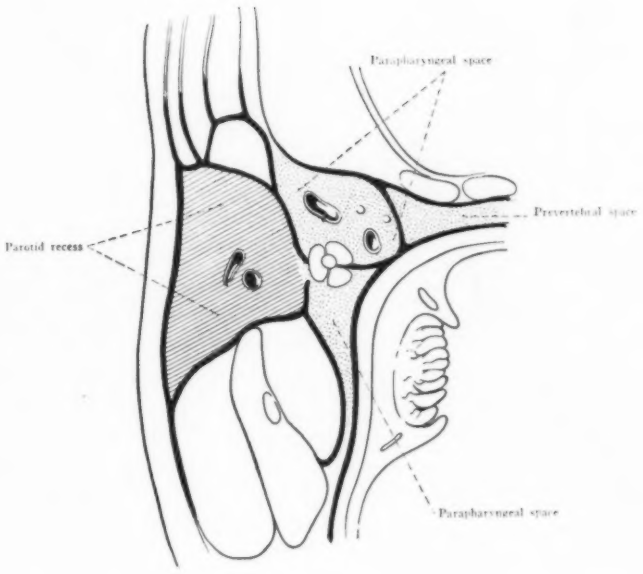

Fig. 4.—Schematized section as in Figure 3 to show pre- and poststyloid compartments of the parapharyngeal space. (From Kirschner, Operative Surgery, Lippincott, with permission)

REPORT OF A CASE

W. B., a 41 year old white male, knew of a tumor in his throat at least five years prior to his seeking medical aid. It had been discovered on a pre-employment physical examination. On a more recent examination, he was urged to have it removed because of apparent increase in size. The only symptom was a vague feeling of fullness in his throat. There was no dysphagia or weight loss or subjective voice change.

Examination, however, revealed the vocal changes usually detected with an oropharyngeal mass. The patient appeared to be in good health and the examination was negative except for a large, firm, nontender, non-pulsating mass which bulged the left side of the pharynx to the midline (Fig. 5). It was apparently the size of a large lemon extending from the region of the eustachian tube to the tip of the

Fig. 5.—Neurofibroma of pharynx.

epiglottis. Palpation of the neck revealed two small anterior and posterior cervical nodes, and a moderate sized bilateral nodular thyroid enlargement. Urinalysis was normal. The blood count revealed a white count of 11,600 with normal differential and no abnormal cells.

The preoperative impression was a mixed salivary gland tumor. Under intratracheal general anesthesia, a vertical incision was made from the region of the eustachian tube by elevation of the palate, posterior to the posterior pillar, to the level of the epiglottis. The incision had to be carried through superior and middle constrictor musculature and pharyngeal aponeurosis to reach the capsule of the tumor. Exposure was obtained by use of Colver forceps and Love retractors. Deep dissection was carried out bluntly. The mass was somewhat adherent superiorly toward the base of the skull. The upper part of the wound was closed with interrupted chromic sutures, the lower part left open for drainage. No pack was inserted into the cavity, since a tracheotomy had not been done. Blood loss during operation was estimated at less than a pint.

During the afternoon and evening of surgery a progressive and rather alarming subcutaneous and mediastinal emphysema developed from the lower eyelids to the lower costal margin, apparently from swallowing air. Careful observation of blood pressure, pulse, and respirations for signs of mediastinal compression was made frequently, but fortunately this failed to develop. Postoperatively, there was a rise of temperature to 101° F. which subsided to normal in four days on

penicillin and streptomycin. Intravenous feedings were used four days, at which time a clear liquid diet was begun. The patient was discharged on the tenth postoperative day.

The fresh specimen (Fig. 2), which was roughly pear shaped, measured 6.5 x 4.5 x 3.5 cm. It presented a smooth glistening surface, pinkish tan and yellow in color. Cut section through the center revealed a cystic cavity which measured 2.5 cm in largest diameter. The wall was somewhat irregular and the cyst contained serosanguinous fluid. Associated with this larger cavity were two smaller ones. The tissue surrounding the cavities was firm and light tan in color. Further sections revealed moderately firm grayish white tissue with softer pinkish tan and pink tissue. Some of it appeared to be gelatinous in nature.

On microscopic examination, the tissue showed an encapsulated connective tissue border. The tumor itself was composed of fibrous tissue. The cells were elongated and had oval nuclei which were either heterogeneously arranged or which formed palisades. In the connective tissue there are zones of edema with myxomatous change, some zones of coagulation necrosis and some areas of hyalinazation. Blood vessels present within the tumor showed similar changes. The pathological diagnosis was neurofibroma of the pharynx (Schwannoma).

On checkup examination three weeks postoperatively, the wound had healed with an imperceptible scar and no neurological deficit could be found. That is, function of the glossopharyngeal, vagus, hypoglossal, and cervical sympathetic nerves appeared to be intact.

SUMMARY

The neurofibroma is a rare tumor of the parapharyngeal space tenting the pharyngeal wall medially to present in the pharynx or laterally to appear in the neck. They may arise from perineural sheath cells of the last four cranial nerves or cervical sympathetic or parasympathetic nerves. However, careful surgical removal may spare injury to these important structures. Routine tracheotomy and external carotid ligation is not always necessary.

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XIV

STRESS AND HYPOADRENOCORTICISM

THE IMPLICATIONS IN OTOLARYNGOLOGY

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Very few specialties in medicine today have not felt the impact of endocrinology and the effects of the rapidly growing list of its therapeutic agents. The tremendous increase in knowledge of the endocrines in the past few years and the terrific upsurge of interest with the advent of cortisone and its derivatives has involved practically every specialty in medicine. Many nose and throat treatments have evolved from this knowledge. Empirical use of cortisone has obscured the basic hypoadrenocortic state which we believe to be the determining factor in many otolaryngologic situations resulting from stress.

It is becoming more apparent that a state of hypometabolism, i.e., hypoadrenocorticism, exists in the general population[1,2,3] to a far greater degree than has heretofore been recognized. Selye and his associates[4] have recently focused attention on the connection between the adrenal gland and otolaryngology.

In previous communications we have elaborated on the detection of hypoadrenocorticism by simple observations which could be corroborated by a minimum of laboratory procedures. We are dealing principally with an inherited deficiency characterized by a particular body build. These patients have a thin asthenic body habitus, sparse body hair, usually an abundant head of fine hair, thin dry skin, long thin necks typical denture formation, long thin non-tapering fingers with the index finger often longer than the fourth finger. The women are frequently flat-chested with extremities proportionately longer than the torso, and often give a history of lowered basal metabolism

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and anemia. The men have sparse chest hair and an absence of hair on the lower lateral two-thirds of the legs. Further findings are susceptibility to bruising, acute costal angle, narrow chest and consequently long thin heart and J-shaped stomach along with a general visceroptosis.

Characteristically, they are usually very meticulous and perfectionistic in all of their activities. Generally the intelligence is above average but they are usually of a nervous, irritable and apprehensive temperament. They often complain of excessive weakness, fatigue, depression and faint easily. A common complaint is inability to fall asleep readily and waking again after a few hours; occasionally complaining of headaches and dizziness. Experience has taught many to resort to food or even alcohol in order to resume sleep. Personality disturbances are frequent including compulsive work habits, inability to relax, depression and sluggishness associated with low blood sugar.

The chief complaints listed for patients with hypoadrenocorticism are often similar to those found in persons who are in a hypoglycemic state. Indeed, a four hour glucose tolerance test reveals enough information to explain most of the symptoms so that further laboratory work beyond a blood count is unnecessary. In younger individuals, we usually find a flat curve with a dropping off between the third and fourth hours. In older individuals, depending upon dietary indiscretions regarding carbohydrates and alcohol, a sudden peak to hyperglycemic levels or a plateau resembling a hepatic curve, both falling to hypoglycemic levels, are generally diagnostic. A routine blood count reveals a relative lymphocytosis and eosinophilia.

Selye in his General Adaptation Syndrome has elaborated on the effect of stress in causing adrenocortical responses, i.e., thymicolymphatic involution, shock and gastro-intestinal ulceration. Any stress elicits increased demands for adrenocortical hormones to meet the increased needs for cellular activity. The 28 known hormones produced by the adrenals are grouped under three separate headings: the glucosteroids, mineralosteroids, and sexogens. Each of these bears its own role in nourishing the tissues, maintaining electrolytic balance, and vasomotor stability. We have shown previously that hypoadrenocortical patients react poorly to stress and actually have demonstrated either an imbalance among the steroid groups or a lowered adrenocortical function. Homeostasis is maintained by proper integration of the two important systems of the human body, the hormonal and the autonomic nervous systems. An involvement of one of these systems necessarily upsets the function of the other. Nervous stimuli may

result therefore in hormonal imbalance or, conversely, hormonal imbalance may be manifest in nervous aberrations such as vasospasm.

Otolaryngologic conditions having an endocrine basis are frequently treated symptomatically. Since it is being recognized that allergic manifestations are on an adrenal cortical deficiency basis, the topical treatment of many ear, nose and throat conditions is minimized by treating the basic dysfunction. Dramatic results are not uncommon. We have repeatedly observed the temporary disappearance of nasal polyps 20 minutes after a single intravenous injection of adrenocortical extract. Prolongation of these effects is obtained by strict adherence to a low carbohydrate, moderate fat and high protein diet.

In all of these conditions adrenocortical extract is given to suppress adrenal cortical hyperstimulation and allow for the regeneration of adrenal cells, or even more specifically, to provide a cortical reserve so that response to stress is not manifested by abnormal vasomotor reactions. It must be emphasized that this is not a replacement form of treatment but an adjunct to the normal physiologic response of the glands to stress.

Dietotherapy is aimed at alleviating the stress imposed by readily absorbable carbohydrates. An autonomic nervous system stabilizer, Bellergal® (composed of belladonna alkaloids, ergotamine tartrate and phenobarbital), is aimed at establishing the normal integration between the nervous and glandular systems, whereas the meprobomates and other tranquilizers block hypothalamic responses. Stressful stimuli are necessary to a normal functioning adrenal cortex.[5] If this current trend of promiscuous dispensing of the tranquilizers continues, Americans of the future will be relegated to a new variety of homo sapiens, characterized by a vestigeal hypothalamus.

There are absolutely no contraindications to the use of the whole physiologically balanced adrenal cortical extract in doses ranging anywhere from 1 to 50 cc. We are at a loss to understand why there is a common impression that cortisone can be used with impunity while the use of ACE is questioned. There is only 1 mgm of cortisone-like activity in 10 cc of adrenocortical extract while the physiologic response is far greater than would be expected from this amount of cortisone because of the synergistic effects of the total steroids present. Electrolytic disturbances are never encountered provided the patient eats a sufficient amount of salt. With ACTH or cortisone, dietary restriction of salt is a necessity because of the tendency to retain sodium.

Originally, in enumerating the symptoms of hypoadrenocorticism, it was realized that many fell within the domain of otolaryngology. These troublesome, aggravating symptoms frequently bring the patient directly to the otolaryngologist. Therefore he should be able to recognize and treat these conditions. Since the steroids are included in his armamentarium at the present time, the whole adrenal cortical extract should have priority due to its safety factors. One objection to the use of ACE is its expense. However, when the ultimate outcome of the case is considered, the over-all cost to the patient is much greater with the individual steroids, for prolonged careful supervision is needed in their use and the frequency of the recurrence of the condition is regrettable. The ease of administration without side effects of the whole extract adds to the gratification of all concerned.

SURGERY

In reports of many surgeons on their operative fatalities it is obvious that many of these patients although diagnosed as status thymicolymphaticus, cardiac arrest, or anesthetic accident, are obviously cases in point. These are what are termed poor surgical risks. On occasion, we have found fasting blood sugars of 20 to 40 mgm per cc. Imagine what would happen to these patients when subjected to general anesthesia for an hour or two? They should be premedicated, provisions made for intravenous infusions during surgery and postoperative intravenous injections of aqueous extract given to forestall shock and insure a smoother recovery. The dose of Eschatin® the aqueous adrenal cortex extract, in all of these situations is 10 cc, or a multiple thereof, depending upon the severity of the condition. Any surgery or stress calls for a marked increase in the supply of adrenocortical hormones to the tissues. Inability of the adrenal cortex to respond to these demands results in a serious pathophysiologic situation.

INFECTIOUS MONONUCLEOSIS

The incidence of infectious mononucleosis seems to be increasing and therefore it is of importance to recognize that there is some specific relationship to hypoadrenocorticism. The presenting symptoms are often ulcerative tonsillar and pharyngeal lesions but on further examination other signs of adrenal involvement are elicited. The use of ACTH in this disease has been reported many times, but in our limited series the response with adrenal cortex extracts has been superior to that of the corticotropin. The basic etiology of this disease has yet

to be determined but all signs and symptoms point to a definite involvement in adrenal cortical function which may account for the prolonged debilitation. In every instance have we noted the characteristic constitutional makeup indicative of hypoadrenocorticism. Treatment consists of injections of 3 to 5 cc of Eschatin every four hours for the first two days, then at increased intervals as indicated. The temperature almost immediately falls to normal, splenomegaly subsides within three days, hepatomegaly and liver tenderness are no longer present after five days. Recurrences have not been encountered. However, the continuation of the diet is emphasized to obviate any physiologic stress on the adrenal cortex.

VIRUS INFECTIONS

The spurious use of sulfonamides and antibiotics has accounted for a greater number of virulent infections presumably due to viral causative agents. In addition to the respiratory symptoms nosebleeds, lassitude, extreme tiredness, depression, et cetera, are found in the general population following these infections but particularly aggravated in the hypoadrenocortic patient. The most aggravating complaint is the persistence of symptoms which do not respond to the antibiotics. In contrast, response is quite gratifying with combined hormonal and antihistaminic therapy. There remains little doubt that the adrenals are intimately related to all virus infections. Kief[6] found morphological evidence of altered histological findings following grippe and pneumonia. Colucci[7] with controlled laboratory studies demonstrated definite signs of hypofunction particularly in cases exhibiting marked asthenia. The adrenocortical hormone of choice is the oily solution, Lipo-Adrenal Cortex, since its action is prolonged. One cc intramuscularly in many instances was found sufficient to obtain recovery.

MÉNIÈRE'S DISEASE

In our original observations on the hypoadrenocortical state we noticed that many patients complained of tinnitus (3 per cent), vertigo (20 per cent), anorexia (34 per cent) and epigastric distress (51 per cent). Ménière's disease has been previously frequently diagnosed in these patients. On treatment of the glandular condition these symptoms were alleviated almost completely, but often with recurrences under severe episodes of stress. Our interest in Ménière's disease was then stimulated to treat patients primarily diagnosed as having this condition. Again the results were uniformly good.

Hilger[8] believes that the symptoms are due to autonomic dysfunction in the labyrinthine neurovascular sphere. The vascular anatomy of the labyrinth is ill-designed for the resorption of the transudate resulting from ischemic damage to the capillary due to angiospasm. Williams,[9] in discussing the etiology of Ménière's disease, mentions that a "stereo-type disorder of the peripheral capillary bed is present in patients with Ménière's disease and other vasomotor neuroses," and that "this evidence suggests that Ménière's disease is one of a group of conditions called variously by different authors—the fatigue phase of the general adaptation syndrome, autonomic disintegrations," et cetera.

Susceptibility to bruising is a characteristic in hypoadrenocorticism because of increased capillary fragility. Menkin[10] demonstrated that adrenal hormones decrease capillary permeability and normalize it. Adjunctively, in this respect a combination of ascorbic acid plus bioflavonoids showing a similar activity has been used successfully in protecting the capillary system against damage.[11]

Angiospasm is caused by stimulation of the autonomic nervous system resulting from hypoglycemia or a gonadal deficiency. One of the principal goals is to correct the erratic blood sugar levels and to supply estrogens or androgens as indicated. Adrenal cortical extract is given intravenously at frequent intervals depending on the severity of the condition. Adjunctive treatment with Bellergal,® Benadryl® and C. V. P.® is often found helpful. It is extremely important to adhere very strictly to the diet.

Other otolaryngologic conditions including vasomotor rhinitis and allergies are often manifestations of the hypoadrenocortical syndrome. With the correction of the steroid deficiency the lymphocytes are broken down to release antibodies which in turn neutralize the specific antigens causing the allergy. Spasm of the trapezius and sternomastoid muscles leads to dull, throbbing, persistent frontal or occipital headaches. These so-called "tension headaches" invariably respond to the basic treatment of this condition.

SUMMARY

The application of hormonal principles to virus infections, vasomotor rhinitis, allergies, certain types of headaches, Ménière's disease, infectious mononucleosis and surgery are reviewed. Supplemental adrenal cortical extract enables these patients to withstand the alarm reaction and stage of resistance incident to surgical stress. Through

the use of adrenal cortical extract, an autonomic nervous system stabilizer and an antihypoglycemic diet, the symptoms of vasomotor rhinitis, the sequelae of virus infections and some types of headaches are alleviated. Many patients complaining of nausea, vertigo, tinnitus, decreased hearing, et cetera, diagnosed as having Ménière's disease, have shown encouraging response under this form of therapy. Theoretically, we believe that all types of Ménière's disease have a common capillary dysfunction due to hypoadrenocorticism. In actuality, these cases have responded so well to treatment of the basic hypoadrenocortical state that further experience may show Ménière's disease to be a localized expression of lowered adrenal cortical function, and infectious mononucleosis, on the other hand, to be a generalized expression of an acutely lowered adrenal cortical function.

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XV

BEETHOVEN'S DEAFNESS

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Probably no other figure in history bore such a fascination for his contemporaries and his biographers alike as Ludwig van Beethoven. There is bequeathed to us a legacy of literature arguing also that there is no other great man in history of whom more is known and of whom so much of what is known is provocative. The controversy surrounding Beethoven's personal tragedy is mountainous because that tragedy was truly Promethean. Much of the greatest music which ever lifted the human spirit flowed from his pen and the most magnificent of this music Beethoven never heard.

The cause of this deafness has been the center of controversy for more than a century. Although the various causes proposed have been largely conjectural, it may be that a reappraisal of a significant portion of the autopsy findings together with other pertinent historical data will shed new light on the specific cause of Beethoven's deafness.

NATURAL HISTORY OF BEETHOVEN'S DEAFNESS

Beethoven's deafness, if we interpret the voluminous records correctly, was slowly and inexorably progressive. It began at about the age of 29, and Beethoven himself never took note of a single remission. By the age of 48, conversation was impossible; he was totally dependent for speech reception upon his celebrated "conversation books." During the last eight years of his life there was no convincing evidence that he heard a single sound. Incredibly, it was in these silent years that the monumental *Ninth Symphony* and *Missa Solemnis* were composed, and *Fidelio* was completed. Total deafness marked his most fruitful years.

As to the origin of his deafness, we have two accounts, both second-hand. Alexander Thayer in his classic biography quotes the

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English pianist Charles Neate, who states Beethoven himself gave him, in 1815, this account: "I was once busy writing an opera. . . . I had a very ill-tempered, troublesome *primo tenore* to deal with. I had already written two grand airs to the same text, with which he was dissatisfied, and now a third, which, upon trial, he seemed to approve and took away with him. . . . I heard a knock at my door, which I at once recognized as that of my *primo tenore*. I sprang up from my table under such an excitement of rage that, as the man entered the room, I threw myself upon the floor as they do upon the stage (here Beethoven spread out his arms and made a gesture of illustration) coming down upon my hands. When I arose I found myself deaf and have been so ever since."[1]

The second account, which Thayer discounts, is given in the so-call Fischoff manuscript: "In the year 1796, Beethoven, on a hot summer day, came greatly overheated to his home, threw open doors and windows, disrobed down to his trousers and cooled himself in a draft at the open window. The consequence was a dangerous sickness, which, on his convalescence, settled in his organs of hearing, and from this time his deafness steadily increased."[2] The date at least is inconsistent with information we have by Beethoven's own hand.

The first reference we have in his words concerning his deafness is contained in a letter to Pastor Amenda, musician, preacher, and lifelong intimate friend, dated June 1, 1801: "Know that my noblest faculty, my hearing, has greatly deteriorated. When you were still with me I felt the symptoms but kept silent; now it is continually growing worse, and whether or not a cure is possible has become a question; but it is said to be due to my bowels (Beethoven was plagued with nearly lifelong prolonged episodes of severe diarrhea). . . . My affliction causes me the least trouble in playing and composing, the most in association with others. . . ."[3]

He elaborates on his deafness in a letter written four weeks later to Dr. Wegeler, a close friend and physicion: "My hearing has grown steadily worse for three years. . . . Frank (Director of the General Hospital at Vienna) strove to restore my hearing by oil of almonds; but alas! my hearing became worse. . . . I was often in despair. . . . Then some medical ass recommended me cold baths, but a more judicious doctor the tepid ones of the Danube, which did wonders; my bowels improved but my hearing remained the same, or in fact rather got worse. . . . I certainly do feel better, but my ears whistle and buzz (*sausen und brausen*) continually, day and night. . . . In any other

profession this might be more tolerable, but in mine such a condition is truly frightful. . . . To give you some idea of my extraordinary deafness, I must tell you that in the theater I am obliged to lean close up against the orchestra in order to understand the actors, and when a little way off I hear none of the high notes of instruments or singers, and if I be but a little farther away I do not hear at all. Frequently, I can hear the tones of a low conversation, but not the words, and as soon as anybody shouts it is intolerable."[4]

During the next sixteen months his deafness evidently reached a level below that of social adequacy. This period was marked by alternating panic and despair as he rushed from physician to physician, trying this remedy and contemplating that one: "For several months," he relates in a letter to Wegeler dated November 16, 1801, "Vering has had vesicatories placed on both arms." Hope springs eternal. "It is true," he continues, "I cannot deny that the ringing and sounding in my ears has become less than usual, especially in the left ear where my deafness began; but my hearing has not improved and I dare not say it has not grown worse rather than better. . . . I am beginning to use the herbs on the belly as suggested by you. . . . Vering will hear nothing of plunge baths, and I am thoroughly dissatisfied with him. . . . What do you think of Schmidt? . . . Miracles are told of *Galvanism*: what have you to say about it? A doctor told me he had seen a deaf and dumb child recover his hearing (in Berlin) again—and a man who had been deaf seven years got well."[5]

His deepest despair was reached at Heiligenstadt where he journeyed to undergo treatment in the fall of 1802. It was here he wrote his celebrated will, in which he rails against his fate and utterly abandons hope. Sorsby[6] quotes Romain Rolland: "It is a cry of revolt and of heart-rending pain—one cannot hear it but be shaken with pity. He is ready to end his life; only moral rectitude keeps him back." The reader must peruse this document for himself; to separate any passage from the whole would be literary abortion.

However, this period passed into one of relative tranquility. He could still hear loud conversational speech (Reis).[7] There is no further mention of his deafness until 1810, when in a letter to Wegeler he states: "Still, I should not only be happy, but the happiest of men, if a demon had not taken up his settled abode in my ears."[8]

Beethoven's fame as a composer had spread throughout Europe. His works were selling so well he had few monetary concerns. Deafness progressed so slowly that we learn of it only through the eyes of

others during the years 1802-1814. Spohr, contemporary composer, noted in 1812 that "it was a difficult task to make him understand, as one had to shout so loudly that it could be heard three rooms distant."[9] Two years later, in 1814, Spohr had occasion to hear Beethoven perform on the pianoforte. He listened aghast to this work, Opus 97 in B-flat major, and notes "it was not a treat, for, in the first place, the pianoforte was badly out of tune, which Beethoven minded little, since he did not hear it. . . . In *forte* passages the poor deaf man pounded on the keys till the strings jangled, and in *piano* he played so softly that whole groups of tones were omitted, so that the music was unintelligible. . . . I was deeply saddened at so hard a fate."[10] How much more sad must Beethoven have been, this once celebrated pianoforte virtuoso who, bowing to his handicap, never after that summer played before the public.

By 1814, Beethoven was using an ear-trumpet, several having been made for him by Johann Malzel, contemporary musician and court mechanician. A headband designed to fix the trumpet (Fig. 1) to his ear, leaving both hands free, probably attests to the fact that he utilized the trumpets primarily in composing. He had eagerly awaited their manufacture—"The ear-trumpets came at last, but were not of the service to me that I expected."[11] Small wonder, considering his high-grade discrimination loss. Beethoven used them for a number of years, nonetheless.

By 1818, according to Schindler, conversation even with the aid of the trumpets became impossible. Sorsby finds evidence that by 1819 "he is spoken of as totally deaf by Zeltzer, who writes to that effect to Goethe, and by Atterbohm: 'He is now what is called stone-deaf.' "[12] He continued to conduct his works, however, until 1822, when he abandoned an attempt to direct a performance of *Fidelio* when it became obvious even to him that he could do no better than hinder the performance.

The composer did have recurrent otalgia but probably not severe pain, because he mentions it only once in his letters, in February 1822: "Last night I was again attacked by earache from which I generally suffer at this season of the year."[13] Carl Czerny, a contemporary, writes to Otto Jahn: ". . . he had suffered from pains in his ears and the like ever since 1800."[14] Nowhere is there to be found any mention of otorrhea.

By 1824, three years before his death, an incident occurred which demonstrated that Beethoven dwelt in total silence. At the conclusion

Fig. 1.—Various primitive ear-trumpets devised for Beethoven by Herr Mälzel in 1814.

of the first performance of his *Ninth Symphony*, the audience broke out in tumultuous applause but Beethoven, whose back was turned to the cheering crowd, was oblivious to it until one of the singers made him turn around.[15]

OTHER ILLNESSES

Beethoven was plagued with chronic ill health from the time of young adulthood. Chief among his non-aural maladies was a con-

tinually recurrent severe diarrhea usually accompanied by colic, and several times, notably in 1812, leading to prostration. He suffered recurrent severe headaches. Attacks of asthma caused him concern that he might have tuberculosis, from which his mother had died.[16] Beethoven was 17 at the time. All his biographers bear witness to the evidence of smallpox over his face, lesions which he bore from early youth. In a letter to Dr. Braunhofer, May 13, 1825, we learn of an episode of hemoptysis and several months of repeated epistaxis.[17] In 1821, he complains of a protracted episode of jaundice, "a most loathsome malady."[18] During the next six years, his liver disease advanced. The cause of his cirrhosis was undoubtedly nutritional, a combination of his well-known desultory eating habits and his severe bowel disorder. He also had a fondness for wine, complaining bitterly to one physician who denied it him in favor of "that deleterious beer which is quite detestable."[19] His final illness began with another attack of jaundice. During a four-month period he developed ascites severe enough to require four paracenteses. In the first, eleven liters were removed and in the second, twenty liters. Hepatic failure was slowly progressive. The proximal cause of death was undoubtedly a terminal pneumonitis. He died on March 26, 1827.

THE CASE FOR SYPHILIS

Theodore von Frimmel[20] and Leo Jacobsohn[21] attribute both the deafness and the cirrhosis to syphilis, claiming that it was common knowledge in Beethoven's time that the master had venereal disease. Sorsby[22] disparages this view, pointing out that this was the cirrhosis of poor nutrition and the evidence for syphilis at all is grossly circumstantial.

In the years succeeding his death, the legend seems to have gained currency that, as befitting the composer of the *Mass in C* and *Missa Solemnis*, Beethoven had always been above the more obvious frailties of the flesh. It is in precisely the matter of sexual morality that the Beethoven legend does not correspond with reality. Reis, a close friend, states so far as Beethoven never having been in love, he was practically never out of it.[23] Wegeler relates to us, "He was frequently in love, but mostly only for a short while each time. Once when I teased him about the conquest of a certain lovely lady, he confessed that she had held him captive longer and closer than any of the others—a full seven months."[24] His friend Friedrich Wähner assures us that "just as he withstood like a man the temptations of wine, so he seems never to have been seduced by the power of love."[25] Actually, nothing could be

further from the truth. Dr. Bertolini, friend and physician from 1806 to 1816, states flatly that he generally had a "flame," and that in addition he was also given to flirtations "from which he did not always emerge happily."[26] Even Thayer, the last man in the world to wish to dwell upon the failings of his hero, comments significantly upon Dr. Aloys Weissenbach's remonstrances as to the state of his morals being absolutely pure, "which, unfortunately, is not true."[27]

Thayer, however, had in his possession, pieces of evidence he could not bring himself to make public. Sir George Grove, on the strength of a physician friend's statement, attributes his autopsy findings to syphilis, and adds in a footnote: "This diagnosis . . . is confirmed by the existence of two prescriptions of which, since this passage in the text was written, I have been told by Mr. Thayer, who heard them from Dr. Bertolini." Ernest Newman[29] tells us "it has been a matter of common knowledge for some time that Thayer had evidence on the subject that he did not think it advisable to publish, and there can be little doubt that his main source of information was Bertolini." Thayer volunteers a significant bit of information: "Through a caprice of Beethoven his cordial relations with Dr. Bertolini came to an abrupt end about 1815; but the doctor, though pained and mortified, retains his respect and veneration for his former friend to the last. In 1831 he gave singular proof of his delicate regard for Beethoven's reputation; supposing himself to be at the point of death from cholera, and being too feeble to examine his large collection of the composer's letters and notes to him, he ordered them all to be burned because a few were not of a nature to be risked in careless hands."[30]

Waldemar Schweischeimer, a German physician, came to the conclusion in 1922 that Beethoven's deafness followed upon an attack of typhus and was related to his gastrointestinal disease, demanding proof that Beethoven ever suffered from syphilis.[31] This unrealistic approach amounts to nothing more than a denial of the possibility of syphilis through lack of positive evidence. It ignores the crucial fact that evidence was withheld from publication because of an exceptional and commendable feeling of piety towards the great master. Bertolini did not die in the cholera epidemic of 1831. He lived until well into the fifties, and Thayer knew him well. Thayer's carefully chosen words concerning Bertolini's "delicate regard for Beethoven's reputation" are decisive. Why should a physician, believing himself to be at the point of death, be grossly concerned over any documents bearing witness to a disease so clearly above moral reproach as typhus or deafness?

Newman quotes von Frimmel's loosely veiled implication: "In addition to possible predisposing causes for his deafness such as measles and scarlatina, there was another that might be assumed, about which I must not keep silence, since many years ago Thayer gave me, by letter, definite information about this *other* malady of Beethoven's."[32] Jacobsohn then comments upon this: "After Frimmel's communication I do not feel I am committing any indiscretion when I say that in the private possession of a man of culture in Berlin there is an as yet unpublished note in Beethoven's own hand referring to a cure that leaves no doubt as to the specific nature of his malady."[33]

As to Beethoven being above now and again placing himself in the occasion of contraction of the disease, Thayer leaves little doubt: "Spending his whole life in a state of society in which the vow of celibacy was by no means a vow of chastity; in which the parentage of a cardinal's or archbishops' children was neither a secret nor a disgrace; in which the illegitimate offspring of princes and magnates were proud of their descent and formed upon it well-grounded hopes of advancement and success in life; in which the moderate gratification of the sexual was no more discountenanced than the satisfying of any other natural appetite—it is nonsense to suppose that, under such circumstances, Beethoven could have puritanic scruples on that point. Those who have had occasion to and opportunity to ascertain the facts know that he had not, and are also aware that he did not always escape the common penalties of transgressing the laws of strict purity."[34]

AUTOPSY FINDINGS

On March 28, 1827, two days after Beethoven's death, Johann Wagner and his assistant, Carl von Rokitansky, performed a careful and exhaustive autopsy. Sorsby's translation of the post-mortem report deserves full quotation:

"The cadaver, and especially the limbs, were very wasted and covered with black petechiae; the abdomen very much swollen and tense with water.

"The auricular cartilage was large and irregularly formed; the scaphoid fossa, but particularly its conchal portion, was very roomy and deeper by half the normal; the various projections and depressions were considerably exaggerated. The external auditory canal appeared to be lined with glistening cutaneous scales; this was especially marked near the concealed tympanic membrane. The eustachian tube was

very thickened and its mucosa rugose and rather narrowed at the bony part. At its pharyngeal opening, and near the tonsils, scarred depressions were to be seen. The mastoid process was large and not marked off by any groove; its cells were conspicuous and lined by a highly vascular mucosa. The whole of the petrous portion of temporal bone, showing large vessels, was similarly well supplied with blood, especially in the region of the cochlea, the spiral lamina of which seemed slightly reddish.

"The facial nerves were of considerable thickness. The auditory nerves on the other hand seemed shrunken and without pith; the accompanying arteries were of the calibre of a crow-quill, and of cartilaginous consistency. The left auditory nerve, much the thinner, arose by three very thin, greyish roots; the right by one root, stronger and pale white. The superficial substance surrounding the fourth ventricle was much denser in consistency and more vascular than these nerves which arose from it. The brain was softer and more watery than normal, and the convolutions appeared to be twice as deep and more numerous than usual. The vault of the skull was markedly and uniformly dense and about half an inch thick.

"The thoracic cavity and its contents were normal.

"The abdominal cavity contained four mass (8 liters) of turbid, greyish-brown fluid. The liver appeared shrunken to half its size, leathery in consistency and greyish-blue in color; its tuberculated superficial surface and its cut surface was full of knotted masses of the size of a bean; all the vessels were very contracted, thickened, and devoid of blood.

"The bile was a darkish-brown fluid containing a gritty sediment, the spleen was twice its normal size, blackish in color, and tough; similarly the pancreas was large and firm, and its duct was the width of a grey quill. The stomach and intestines were very distended with gas.

"Both kidneys were contained in a cellular membrane, one inch in thickness, dripping a turbid brownish fluid, its structure was loose, and pale red in color; each calyx contained a chalky deposit, wart-like in shape, and similar to a pea cut across."[35]

THE NATURE OF HIS DEAFNESS

Beethoven states in the "Heiligenstadt Will" that prior to the onset of his deafness in his late twenties, he had exceptionally keen

hearing. The first clue to oncoming deafness was the loss of high-tone perception—he could not hear the high-pitched instruments or voices on stage; he could not hear a shepherd's flute when it was plainly audible to a companion. Initial high-tone loss is not perfectly characteristic of either otosclerosis or cochlear deafness. The course of his hearing loss, that is, beginning in the high tones and progressing slowly to practical extinction, is characteristic of some type of perceptive deafness. It seems unlikely that Beethoven's ears recruited or were the seat of diplacusis or distortion. To possess these characteristics and still love to conduct a full orchestra, seated at his pianoforte bathed in probably 80 to 100 decibels of sound, would be a feat insuperable for even such a giant as Beethoven. This, together with the absence of any recorded vertiginous episodes, makes cochlear deafness or Ménière's disease improbable. Its progressive, absolutely unremitting nature is against hysterical deafness.

No evidence suggesting conductive deafness was found at autopsy. Johann Wagner, of the Vienna Pathological Museum, treated Beethoven at one time and must have been keenly interested in the cause of his deafness, judging by the length at which he dwelt upon the auditory apparatus at necropsy. There is no mention of otorrhea during Beethoven's lifetime, although the reports of recurrent otalgia are puzzling. Wagner's description of a large cellular mastoid is not consistent with arrested mastoid development seen with chronic middle ear infections; it is possible that the otalgia was due to external otitis, in view of the indicated presence of a fine scale lining the external canals, and the noticeable absence of any mention of cerumen. This would not be unreasonable considering the vast amount of materia medica Beethoven must have poured into his ears at the behest of his myriad of medical advisers.

Otosclerosis has been advanced as a cause of his deafness. Wagner and Rokitansky were careful and painstaking workers; it is doubtful that such a major flaw as stapedial footplate fixation would have escaped their notice in their examination of the cochlea. Furthermore, pure otosclerosis, even in its advanced form, does not produce total loss of hearing or neural atrophy. Proponents of this theory point out a singular piece of evidence—the use of a drumstick (*baguette de bois*) by which Beethoven listened to the pianoforte while composing, "one extremity of which he applied to the piano and the other he held between his teeth."[36] Great notoriety has been afforded this device as an early primitive form of bone-conduction hearing aid, but in actuality it deserves little credence. The sole source of information

is an obscure French physician named J.A.A. Rattel, who could not have observed it first-hand because he was not a contemporary of Beethoven's. The master may have experimented with it briefly but it could not have been his practice, since it totally escapes mention by his multitude of biographers and correspondents. Were it so, it is suggestive of conductive deafness but far from definitive evidence: the sound-gathering properties of his tiny ear-trumpets would certainly be less than that of a rigid instrument transmitting vibrations from the piano-frame directly to his skull. Indeed, teachers of profoundly deafened children use this same principle in demonstrating the nature of sound; furthermore, they bypass the drumstick, the child applying his teeth directly to the edge of the piano top.

Of the choices outlined, the most likely seems a perceptive deafness, the basis of which was either a progressive neuroepithelial or neuronal atrophy.

THE CAUSE OF HIS DEAFNESS

Excluding otosclerosis and other forms of middle ear deafness and endolymphatic hydrops, there remains eighth nerve deafness and central deafness to be considered. Central deafness may easily be discredited; even late in his disease, Beethoven had no difficulty interpreting sounds once they could be gotten into the brain stem.

Eighth nerve deafness then seems to be the most likely possibility. Various authors have described two types of eighth nerve deafness, viz., epithelial and neural.[37] The marked eighth nerve atrophy found at necropsy militates for the latter. Usually there is some degree of recruitment when the former type obtains, and it has been shown improbable that Beethoven demonstrated any recruitment. It is possible that the two conditions coexisted, but it is not considered good medicine to make two diagnoses where one will suffice.

The causes of eighth nerve atrophy are legion, the vast majority falling in that unfortunate medical catch-all, idiopathic. Since, however, we know a great deal of Beethoven's medical background, a number of specific possibilities may be explored.

Variola, of which Beethoven bore scars, is not associated with eighth nerve deafness. Typhus was endemic in Vienna at the time, and this was probably the "dangerous sickness" which Beethoven contract-

ed in 1796. The deafness of typhus is often more apparent than real; it varies from day to day according to the severity of coma and disorientation, and improves rapidly if the patient recovers.[38] The deafness of a high febrile infection is severe but upon lysis does not progress, the fever having done its damage. The course of Beethoven's deafness is similar to the inexplicable progressive perceptive deafness of adolescence except for the age factor.

Indeed, one might be forced into agreement with Schweisheimer and Sorsby that this is simply a chronic progressive nerve deafness without definable cause, of the type seen frequently even today, were it not for a significant portion of the necropsy report which has escaped comment. The "superficial substance surrounding the fourth ventricle," which can refer only to piarachnoid of the brain stem, "was much denser in consistency and more vascular than these nerves which arose from it." Granting the advanced atrophic state of the eighth nerves exiting here, such a contrast, considering the normal cobwebby appearance of the piarachnoid, can be taken only to refer to a marked thickening of the meninges in this area. This, together with the increased meningeal vascularity and the absence of any record of past acute meningitis, would tend to suggest a chronic, low-grade, proliferative meningitis.

It is possible that such meningitis was the proximal cause of his deafness. Tuberculous meningitis produces cranial nerve palsy, probably by pressure, about the base of the nerve at its exit from the brain stem, producing neural atrophy. The course of tuberculous meningitis is severe and ravaging; no such process afflicted Beethoven. The only other chronic infectious granuloma which is implicated in Beethoven's past is syphilis. This disease can produce a cranial nerve palsy by pressure exerted upon the nerve by organized exudate about the brain stem. An example of this (Fig. 2) has been observed at the University of Michigan in which the cause of a clinical tenth nerve palsy was found at necropsy. The second overlooked portion of Wagner's and Rokitansky's careful necropsy involved their description of the internal auditory arteries. These arteries "were of the calibre of a crow-quill, and of cartilaginous consistency." The normal artery has the diameter of paper-clip wire and is of silken consistency. These vessels, then, undoubtedly partook in the disease process. Syphilis, wherever it exists, may be considered a disease of blood vessels.[39] There is agreement among pathologists that syphilitic leptomeningitis rarely, if ever, exists without accompanying arteritis. It is most marked about the base of the brain, with early involvement of the intracranial portions

Fig. 2.—Example of organized syphilitic exudate about the brain stem producing a bilateral tenth nerve palsy. (Courtesy Dr. A. C. Furstenberg)

of cerebral nerves, and may exist for a long time with few symptoms.[40,41]

It would seem the only reasonable arguments against the implication of syphilis would be twofold: 1) the unavailability of temporal bone histologic findings and suitable serologic tests, and 2) the absence of other syphilitic stigmata in either Beethoven's medical history or autopsy report. The former are, of course, highly desirable and, in our day, usually prerequisite to final diagnosis. It is equally true, in spite of the deplorable popularity this diagnosis had years ago, that the astute clinician was usually able to recognize the disease without benefit of either histologic or serologic evidence. Other syphilitic stigmata need not be present in syphilitic deafness, as has been pointed out. Ciotto has shown in his classic monograph that the diagnosis of neurosyphilis based upon eighth nerve involvement alone is unjustified; it is more common for there to be no other central nervous system signs.[42]

SUMMARY

The natural history of Beethoven's deafness, together with a judicious examination of pertinent necropsy findings, provides adequate evidence for a chronic, progressive perceptive deafness. Marked eighth nerve atrophy was present, and most recent authors have considered this idiopathic. Recorded gross pathologic findings, to which it would seem too little attention has been paid, indicate a chronic granulomatous inflammation producing a basilar piarachnoiditis and eighth nerve arteritis, capable of eventuating in auditory neural atrophy. Syphilis is the most likely cause of this low grade meningitis which existed without other central nervous system signs.

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XVI

NEUROGENIC TUMORS OF THE NOSE

A REPORT OF TWO CASES

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Congenital neoplasms of the nose are rare. They have been divided into three groups:[18] 1) tumors of neurogenic origin, 2) tumors of ectodermal origin (dermoid cysts) and 3) tumors of mesodermic origin (hemangiomas). Out of these, the neurogenic ones form an interesting group of tumors because of the controversy regarding the origin of the neurogenic element. The two cases reported here are unusual and interesting because in addition to mature neuroglial tissue they also show the presence of striated muscle fibers.

REPORT OF CASES

CASE 1. A female child aged three years was admitted with a polypoidal growth arising from the septum of the nose since birth. The growth had gradually increased in size. A clinical diagnosis of septal dermoid was made and the tumor was excised. The specimen consisted of three pieces of tissues each measuring nearly one centimeter in diameter, firm and greyish in color. The cut surface was uniformly grey, moist and gelatinous in places.

Histological examination showed the tumor to be composed mainly of fibrous and glial tissue. Large islands of glial tissue were separated by fibrous septa. The glial tissue was almost entirely composed of astrocytes with a fine fibrillar matrix resembling normal nerve tissue (Fig. 1). The astrocytes were mainly pilocytic but at places larger cells with 3 to 4 nuclei resembling the gamestocytic astrocytes were also seen. Groups of small round cells which imperceptibly mixed with the surrounding neuroglial and fibrous tissue were noticed. These small cells had vacuolated cytoplasm, indistinct cell border and

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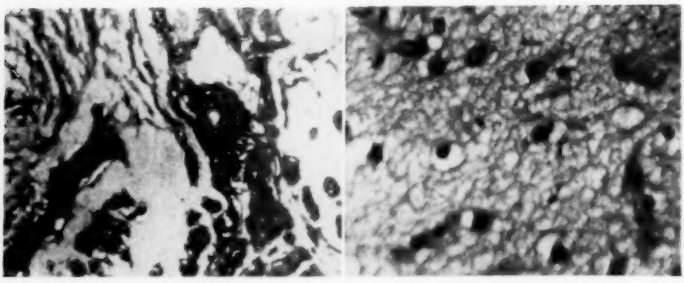

Fig. 1.—Photomicrographs showing large islands of glial tissue separated by fibrous septa. Pilocytic astrocytes shown in the section on the right.

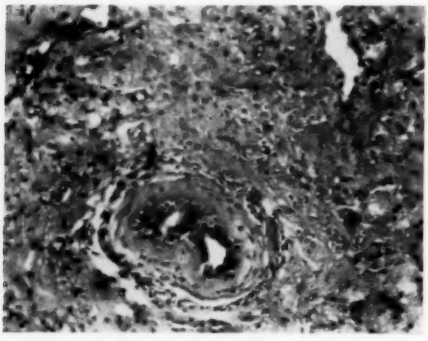

Fig. 2.—Photomicrograph showing the neuroglial tissue in the dermis around the skin appendages. (H. R. x 130)

darkly staining nuclei. Mitotic figures were seen in some of them. These cells cytologically appeared to be primitive or undifferentiated neurogenic cells. There were few blood vessels in the fibrous septa but they contained bundles of striated muscle fibers. With Mallory's P.T.A.H. stain the glial tissue was stained pale purple while the striated muscle fibers were stained a deep blue and showed cross striations (Fig. 3). The presence of the striated muscle fibers was also confirmed by iron hematoxylin and van Gieson's stain. No other tissue was seen in the several sections studied from the tumor.

Fig. 3.—Showing the striated muscle fibers in the fibrous septa. (Mallory's P.T.A.H. Stain x 300)

CASE 2. A female child aged three years was admitted with a swelling on the bridge of the nose since birth which had gradually increased in size. A clinical diagnosis of encephalocele was made and the tumor was excised. The specimen consisted of an oval tumor mass about three centimeters in diameter and covered by healthy skin. The tumor had a firm consistency and a greyish color. The cut surface was uniformly greyish and moist.

Histologically the tumor was similar to Case 1. The dermis was seen to be occupied by neuroglial tissue which surrounded the appendages of the skin (Fig. 2). The epidermis was normal. The groups of small round cells, seen in Case 1, were not present. The neuroglial element was very predominant in this tumor and only some fibrous septa showed a few striated muscle fibers with Mallory's P.T.A.H. stain.

COMMENT

A review of the literature shows that various kinds of neurogenic tumors of the nose (gliomas, neurofibromas, ganglioneuroma, schwannoma,[9] meningioma,[11] encephalocele[10] and meningoencephaloceles,[7] etc.) have been described. Nasal gliomas were first described by Schmidt[14] and since then several cases have been reported in the literature. All these tumors are described as consisting of primitive or well differentiated nerve tissue. In none of these cases was the presence

of muscle fibers described with the exception of Bratton's[4] first case which was situated in the palate and in this case their presence has been explained as being the muscular remnants of normal constituents of the soft palate.

The origin of the neurogenic element of these tumors has been a controversial issue. It has been explained that they are as a matter of fact encephaloceles which have become cut off from their origin.[1,8,14,15,18] The argument in favor of this view is that these tumors consisted entirely of nerve tissue. The absence of nerve cells, which otherwise should be expected in encephaloceles, has been held against such a view.[4] Moreover a true encephalocele should present itself in the neighborhood of the frontonasal process while the nasal gliomas have been found to occur anywhere in the nasal region.[3] It has also been suggested[4] that these tumors are derived from the displaced marginal islands of tissue from the optic plate or the pleuripotential neural ectoderm. Other views suggest their origin from olfactory placodes, olfactory membranes, organ of Loci and organ of Jacobsen. These views have been discussed in detail by Schall and Lineback.[13]

All these views point to a neurogenic origin of these tumors but this concept cannot explain the presence of striated muscle fibers in the two cases reported here. On embryological grounds it seems that these cases should be regarded as teratomas with predominant development of neuroglial element. It is interesting to note that these cases are similar to Bratton's[4] second case in their symptomatology and to a considerable extent, in their histology. In some of the sections in our cases the neuroglial tissue was so overgrown as to simulate nasal glioma and only with Mallory's P.T.A.H. stain could the presence of striated muscle fibers be detected. This observation brings out another possibility that some of the tumors designated as nasal gliomas may be teratomas with one-sided development. The presence of the muscle fibers in Bratton's first case may not be because of its situation in the soft palate, but, as pointed out by Willis,[19] due to the teratomatous nature of the tumor per se.

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XVII

ANTROCHOANAL POLYPS

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Choanal polyps may exist for some time before recognition, and when discovered often are inadequately treated.

Removal of the growth is only the initial step in the management of choanal polyps. These growths have a tendency to recur, and this may take place as long as remnants of the pedicle exist in the nose or the antrum.

Killian[1] 61 years ago traced the pedicle of choanal polyps to the maxillary sinus. His investigations apparently ended at the maxillary ostium, for he failed to report on any intrasinus observations concerning the pedicle or its attachments.

Since then, the stalks of these growths have been traced by various observers through the ostium into the sinus and to the point of origin on the sinus walls.[2-4]

Antral polyps may arise from any wall of the sinus, but the most frequent site of origin is the margin of the ostium.[4] It is understandable that polyps arising at the ostium or nearby may pass readily through the ostium into the nasal cavity and thence to the nasopharynx.

Somewhat more difficult of explanation, however, is the mode of travel from one of the other walls across the cavity to the opening. As Hirsh[4] says, "It is possible to believe that a thin strand arises from the floor of the antrum and makes an aim for the ostium." He offers the explanation that "during a state of excessive edema a portion of the sinus mucosa bulges through the opening and becomes incarcerated in the middle meatus." The stalk is formed as the edema recedes and

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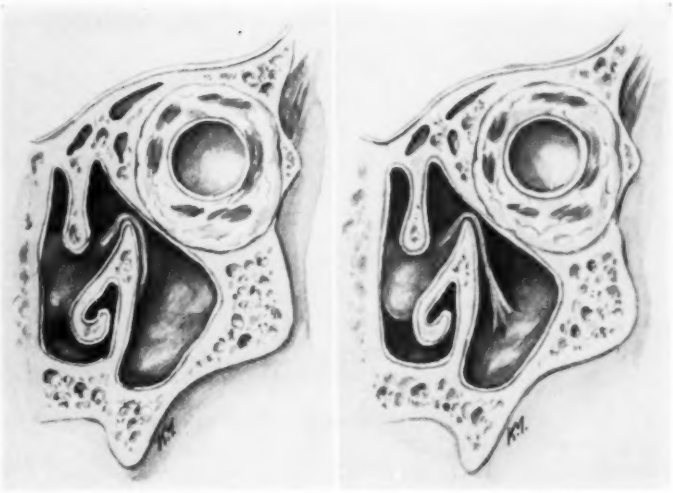

Fig. 1.—Drawings illustrating two of the common points of origin, on the sinus walls, of antro-choanal polyps.

the sinus membranes shrink back to the bony walls, leaving a tubular trail of mucosa to the pinched-off portion in the middle meatus. The portion in the nose, in consequence of passive congestion, gains in size and becomes a solitary nasal polyp with antral connections.

This explanation is more readily accepted when the nature of the antral growth is understood. Many writers,[5] including Killian, have called attention to the cystic character of these growths and it has been noted by the writer on many occasions. He has also noted a rhythmic up and down excursion of the cyst corresponding to the patient's inspiration and expiration. Extension into the nasal cavity occurs as the cyst expands and the pressure builds up to force a portion of the sac through the opening. The pedicle and the polyp thus formed often contain fluid which escapes as the pedicle is severed.

In other cases a firm polyp is found in the antrum attached to a fibrous pedicle, while in some instances the growths are multiple, completely filling the cavity. Cases such as this are usually associated with long standing recurrent polyposis.

No explanation has been forthcoming as to the predilection of these polyps for the nasopharynx. One may assume that these growths,

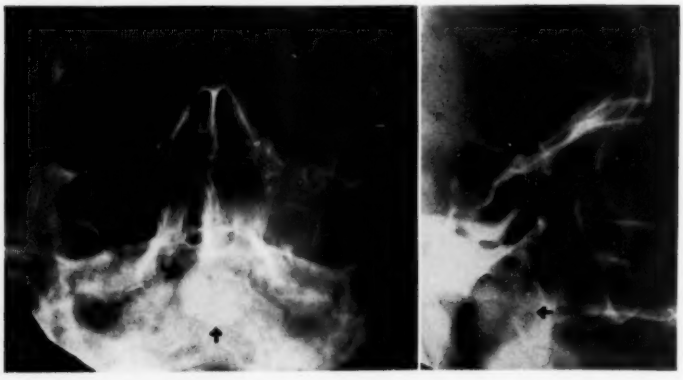

Fig. 2.—Waters and lateral views showing polyp in choana. Left antral opacity is also revealed.

composed as they are of edematous tissue and often containing cystic fluid, elongate readily and by their sheer weight soon reach the floor and pass steadily to the nasopharynx.

Antrochoanal polyps are not limited to any age group. They appear in young children and in elderly individuals, but more frequently in young and middle aged adults.

Characteristic of choanal polyps in children is an occasional anterior prolongation of the polyp which juts off from the main or choanal portion toward the anterior nares. This bilobal characteristic was observed in 1909 by A. Brown Kelly,[3] and one such case is recorded herewith (Case 1).

The pedicle varies considerably in size and appearance. It may be a thick edematous structure or it may be a thin firm stalk, its diameter being determined by the size of the opening through which it passes.

An underlying allergy may be demonstrated in most of the cases of choanal polyps. Rarely there is present a suppurative sinusitis which is scarcely to be considered as a factor in the formation of this type of polyp. The polyp, on the other hand, because of its tendency to block the ostium, could contribute to the persistence of suppurative sinusitis should it develop.

Nasal obstruction may be the lone symptom of choanal polyp. The extent of the blockage depends on the size of the growth and varies from partial unilateral to complete nasal obstruction as the nasopharynx becomes entirely blocked off.

The polyp is best seen by postnasal examination, but occasionally is viewed by anterior rhinoscopy, or it may be revealed by x-ray studies. Postnasal examination is indicated in all cases of nasal obstruction, for not uncommonly this symptom is attributed to nasal allergy or a deviated septum and the growth in the nasopharynx is overlooked.

Proper management includes not only removal of the growth in the nasopharynx but elimination of all factors which may contribute to recurrence. This includes allergy management, removal of growths from the antrum by way of the canine fossa, and finally the establishment of a permanent nasoantral opening in the inferior nasal meatus.

With the discovery of the choanal polyp, x-rays of the sinuses are studied to determine the nature of the antral attachments of the polyp. Many of these may be revealed only with the aid of a contrast medium instilled into the sinus.

REMOVAL OF POLYP

The usual method of removal is with a snare with recovery of the polyp in the pharynx.

Snare Wire Technique. Following topical anesthesia, a nasal snare wire is passed along the floor of the nose into the nasopharynx and around the polyp. If the growth is large and protrudes down below the palate, a long snare wire may be required. This may be manipulated by direct vision and forceps or fingers are used in the pharynx to guide the wire around the polyp. When possible, the wire after passing around the polyp is slipped along the pedicle forward toward the ostium before the pedicle is severed.

This method is not always as easy to carry out as it may seem and may be unpleasant for the patient and frustrating for the operator.

Since the purpose of the snare wire is to sever the pedicle, this may be accomplished in a simpler and more dependable manner by use of a hook without the need of manipulation of the polyp itself.

The original hook technique was described in 1887 by Lange[7] years before the origin and antral connections of these growths were known or suspected. He used an instrument resembling a button hook with which he tore through the pedicle, and either drew the polyp out through the nose or recovered it in the throat, depending upon its size. This method was advocated by Waldapfel,[8] who in 1951 reported its successful application in two cases.

The instrument used by the writer is a hook-shaped knife. It resembles the Lange hook in size and shape, but has a cutting edge which permits the operator to cut cleanly through the pedicle rather than tear it (Fig. 3).

Fig. 3.—Hook knife.

Hook Knife Technique. The knife is passed along the floor to the nasopharynx and placed lateral to the polyp with the hook pointing upward. It is drawn forward until the pedicle is contacted, then pulled with sufficient force to sever the pedicle. The polyp may be recovered in the pharynx, or, if sufficiently small, is drawn out through the nose as the pedicle is severed.

Transantral Technique. In some cases it is advantageous to carry out all surgical procedures in one sitting. In that event the antrum is exposed through the canine fossa, the intrasinus portion of the pedicle is severed and the choanal growth removed through the mouth.

TREATMENT OF THE ANTRUM

In those cases in which the polyp arises from the margins of the maxillary ostium or an accessory ostium no trace of the growth remains in the sinus after removal of the choanal extension and the likelihood of recurrence is slight. In most cases, however, evidence of remaining growths is revealed. Should this fact be established by x-ray examination, the Caldwell-Luc approach is advocated. All growths are removed from the antrum, and a window is placed in the inferior meatal nasoantral wall. The lining membrane of the sinus is not disturbed as a rule. This may seem edematous and irregular

Fig. 4.—(Case 2). Lateral view showing filling defect in left maxillary sinus. Three large polyps, one with stalk, were removed from the upper part of the sinus.

with polypoid areas, but mucosa such as this returns to normal with the establishment of adequate drainage and control of allergy.

REPORT OF CASES

CASE 1. M.F., a nine year old girl, was seen February 10, 1956, at the Illinois Eye and Ear Infirmary with the complaint, "I cannot breathe through my nose." The mother stated that the patient had always had nose trouble. She had frequent colds, snored while sleeping, and was a mouth breather. On April 23, 1956, under local anesthesia, the polyp was removed with a nasal snare. This solitary growth which had completely filled the left nasal cavity consisted not only of a choanal extension, but one reaching the anterior nares as well. A thin pedicle attached to the choanal portion measured 7 mm in length, and apparently had been severed at the ostium.

The pathologist's report: Covered on most of its surface by partially desquamated respiratory epithelium. Stroma is very loose, myxomatous-like fibrous tissue with a moderate number of scattered lymphocytes and plasma cells.

X-ray studies with iodized oil in the left antrum revealed a space-filling defect occupying the central portion. On August 6, 1956, an external antrostomy was carried out. Through a canine fossa approach a large polyp was revealed with a pedicle near the ostium. This was removed and a naso-antral window placed in the inferior meatus. The sinus mucosa appeared to be normal.

On April 2, 1957, nine months later, at our request, the patient returned for examination. The nose appeared to be normal and she was symptom free.

CASE 2. N.D., a 16 year old boy, was seen June 27, 1956, in the Outpatient Department of Illinois Research and Educational Hospital. For three years he had suffered from frequent colds appearing at three-month intervals and lasting three weeks with nasal stuffiness which has been on the increase.

A large solitary polyp presented itself in the left nares near the floor and about half-way back toward the nasopharynx. This was removed along with a pedicle. X-ray films (Fig. 7) revealed a mass in the left antrum, and at operation on August 16, 1956, three large polyps were removed from the sinus. They all were near the roof of the cavity, and one was attached to a stalk which protruded into the nasal cavity through an accessory opening. He since has been under treatment in the Department of Allergy, and has been seen at various times in the Department of Otolaryngology with a minimum of symptoms.

CASE 3. G.H., a white adult male, was first seen in my office December 17, 1953. A large polyp was removed from the right nasopharynx with a snare, and on January 25, 1954, three weeks later, a similar growth was removed. On February 2, one week later, a thin elongated polyp was seen in the nasopharynx on the right side. The snare was passed around it and manipulated forward along the pedicle toward the maxillary ostium before it was severed. The growth, when removed, was 4 cm in length and could well have been the pedicle of the original polyp, for the patient had no further trouble for two years. On April 16, 1956, a return of the growth necessitated its removal, this time accomplished with a hook. X-ray studies were then made

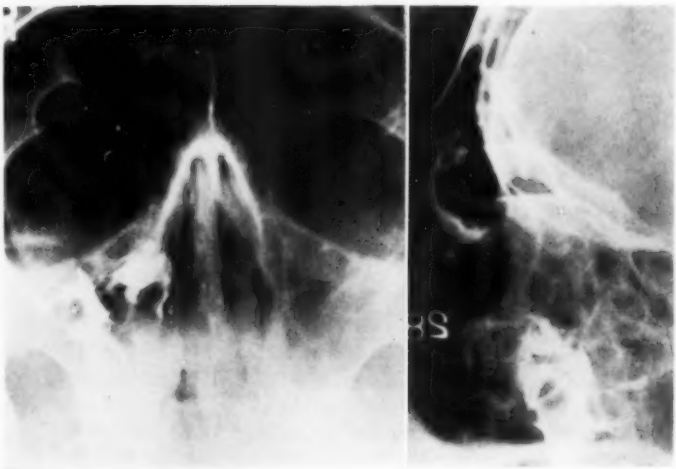

Fig. 5.—(Case 3). Two views showing multiple polyp formation in right antrum in case of recurrent choanal polyps.

and they revealed what appeared to be multiple polyps in the right antrum (Fig. 8). He was urged to have these removed by antrostomy, but refused, and has not been seen since.

CASE 4. H.H., a white male, was seen first on September 11, 1944, complaining of fullness in the ears. Inflation brought about some relief, and he was not seen again until October 4, 1947, when a polyp which was lying low in the right nasal cavity was removed. Two weeks later another growth was removed from the same area. On February 12, 1949, one and one-half years later, another polyp was seen on the floor of the right nasal cavity which extended back to almost reach the nasopharynx. After removal, the patient had considerable relief for a few years, but finally came in again February 3, 1956, seven years later. He complained of a complete nasal blockage and had been breathing through his mouth for several months. The nasopharynx was completely blocked by a huge polyp which extended well below the palate. This growth was removed piecemeal, the lower portion through the mouth by means of a tonsil snare. Other portions were removed with nasal snare through the nose. Six sittings were required over a two week period until finally the method was adopted which should have been used in the first place. The pedicle was

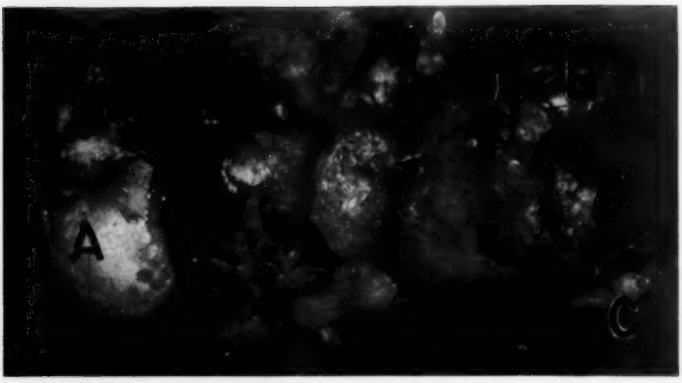

Fig. 6.—(Case 4). Photograph showing massive polyp which extended into pharynx. Polyp, reconstructed after removal piecemeal, measured over 3 inches in length. Portion A, pharyngeal extension; B, extension into left posterior nares; C, the pedicle.

severed with a hook knife, and the large remaining portion was recovered in the mouth.

The entire mass, when reconstructed, measured over 3 inches in length, with a prolongation advancing into the left nasal fossa (Fig. 6).

Five weeks later the patient returned, and then for the first time a study was made of the right antrum. Irrigation produced a purulent exudate and oil was instilled for x-ray study. On April 14, by way of external antrostomy, a polyp with pedicle was removed and a permanent opening made in the naso-antral wall.

On May 28, a small polyp was removed from the right middle meatus which apparently had no connection with the antrum.

One year later, April 30, 1957, he returned for a check-up. He was found to be polyp-free and the antrum was negative on irrigation.

The pathologist's diagnosis was inverting papilloma, and examination of the old slides made from the growths removed in 1947 and 1949 revealed the same type of tissue.

Two other cases of inverting papilloma arising in the antrum and proceeding to the choana have been seen by the writer during the past two years.

CASE 5. J.O., 19 year old white female. Since the age of 12 she had had single polyps removed from the left side of her nose every year or two. In March, 1957, that side of her nose was again blocked and a polyp was seen in the nasopharynx. On July 26, this was removed as an office procedure, using a hook knife. The antrum was then irrigated and oil instilled for x-ray study. Straw-colored fluid escaped during lavage and the films revealed a large space-filling defect occupying most of the sinus.

Within two months another polyp was observed in the left middle meatus. On September 19, the left antrum was opened, revealing a large cyst which filled the sinus. Efforts to remove it intact caused collapse, and then it could be seen that strands of mucosa passed through the ostium to the polyp in the nose. Traction on these strands drew a flattened-out polyp into the antrum through the ostium. No other growths were found in the sinus and the lining mucosa was pale but not polypoid.

SUMMARY AND CONCLUSIONS

Antrochoanal polyps may appear in individuals of any age, including childhood.

The growths may be traced by their pedicles to the maxillary or an accessory ostium, and in the sinus to an elevation on one of the walls. The elevation may be in the form of a polyp or may be a cyst in the edematous mucosa.

The adoption of the hook knife technique greatly facilitates the removal of the choanal growth. By this means the pedicle is severed without the need of manipulation of the polyp itself.

Removal of the choanal growth affords immediate relief, but there is a strong likelihood of recurrence as long as remnants of the pedicle remain in the antral cavity.

The nature of the antral attachments may be revealed by x-ray studies with a contrast medium in the sinus. The presence of a space-filling defect is sufficient indication for an external approach.

This is made through the canine fossa; the pedicle is located when possible, and removed along with its base on the sinus wall. All other polyps and cysts are obliterated and a permanent nasoantral opening is placed in the inferior meatal wall, all this with the least possible disturbance to the sinus mucosa.

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XVIII

TUBERCULOSIS OF THE THYROID GLAND

REPORT OF A CASE

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AND

E. Shanon

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During the course of tuberculosis anywhere in the body nonspecific changes may be observed in the thyroid gland: hyperplasia, in the acute stage, and fibrosis leading to atrophy in the chronic form of the disease. However, specific tuberculous infection of the thyroid gland is uncommon. Rokitanski stated in 1861 that tuberculosis never occurred in the thyroid. For a considerable time the gland was thought immune to that infection. Later, however, Cohnheim described miliary tuberculosis of the thyroid as a part of generalized miliary spread. Chiari, Weigert and others recorded caseous tuberculosis of the thyroid.[1]

The miliary form of tuberculosis of the thyroid is not an infrequent condition. Its recorded incidence would probably increase if routine studies of the thyroid were to be made in all post-mortem examinations on patients who died of tuberculosis. In 1563 autopsies on tuberculous patients Hegar[2] found specific affection of the thyroid gland in 52 cases of miliary tuberculosis, and in five cases of chronic caseous tuberculosis.

In some of the cases reported in the literature[1] the diagnosis of tuberculous thyroiditis does not seem adequately proven. Mere presence of giant cells, lymphocytes, fibrosis and destruction of follicles is not sufficient to establish the diagnosis of tuberculosis. These morphological changes may be characteristic of a chronic, nonspecific thyroiditis with pseudotubercles. The prerequisite for a diagnosis of tuberculosis is the presence of tubercle bacilli or central caseation in the tubercle. The finding of an additional focus elsewhere in the body may lend support to the diagnosis.

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Goldberg[2] stated that a clinical diagnosis of caseous tuberculosis of the thyroid has been made very few times and only in those cases in which a cold abscess or a fistula was present, or in which tubercle bacilli were found in the pus or there was associated active pulmonary tuberculosis or local infection of lymph nodes or larynx. Other authors[3] stressed also that diagnosis prior to operation could seldom be established.

REPORT OF A CASE

A woman, 35 years old, born in Russia, residing in Israel for the last eight years, was admitted to the ear, nose and throat department on September 30, 1956. Her father and his brother died of pulmonary tuberculosis. Her mother, two brothers and two sisters were living and well. In childhood the patient had chicken pox and typhoid fever. In 1933 she had malaria. In 1940 she was ill with pneumonia. In 1948 she developed left pleurisy which lasted for six months. In the same year an appendectomy was done. During the period from 1948 to 1955 repeated x-ray examinations of the chest were done and the patient was told that her lungs were clear. In 1952 she consulted a gynecologist because of sterility; tubal insufflation was done, following which pelvic peritonitis developed. In 1953 biopsy of endometrial tissue revealed tuberculosis. The patient received 150 grams of para-aminosalicylic acid (PAS) and only 12 grams of streptomycin which was discontinued because of vertigo.

The present illness started a year prior to admission, when the patient experienced pain in the neck radiating to the nape and to the left hemicranium. She also had difficulty in breathing in the supine position. Three months prior to admission she felt a hard, tender mass on the left side of her neck, which increased gradually in size. Difficulty in swallowing hard food appeared as well as slight discomfort on turning the head sideways. Throughout the course of her present illness the body temperature was normal. Erythrocyte Sedimentation Rate: 39 mm in the first hour (Westergren). An x-ray of the chest showed no pathological findings.

On admission the patient was in good condition but rather pale. The body temperature was normal. The larynx was normal in appearance and function. The left thyroid lobe was enlarged, hard, tender and it moved on swallowing. The right lobe appeared unaltered. No lymph nodes were palpable in the neck. Examination of the chest, abdomen and extremities showed no pathological changes. Eye grounds were normal. Gynecological examination revealed a cystic mass, the

size of a hen's egg, to the right of the uterus, possibly a tubo-ovarian cyst of tuberculous origin.

Laboratory findings: hemoglobin 10.1 gr per cent; R.B.C. 3,900,000/cmm; W.B.C. 6200/cmm; E.S.R. 6/10 mm. Routine blood chemistry showed normal values. Kahn negative. P.B.I. 3.2 per cent, plasma colloid gold curve negative. No acid fast bacilli were found in the sputum or in the gastric juice. There was a trace of albumin in the urine and a few leukocytes. Urine culture was sterile. B.M.R. —7 per cent. Radioactive iodine uptake (Dr. Z. Lewitus) was 12.5 per cent of the tracer dose after two hours and 24 per cent after 24 hours; 66 per cent of the dose was excreted in the urine during 24 hours. These results are in the lower range of the normal, according to the standards of our laboratory. The profiling of the gland with a collimated directional scintillator showed that most of the tracer was concentrated in the right lobe; the hard left lobe was characterized as "cold."

X-ray examination of the chest revealed several, small, calcified foci in both lungs. A slight indentation could be observed in the cervical part of the left lateral wall of the trachea.

The clinical picture of chronic thyroiditis in conjunction with the history of pulmonary and genital tuberculosis suggested the possibility of a tuberculous thyroiditis.

Following the administration of nicotibine (200 mg daily) and PAS (12 grams daily) for one week, as a precautionary measure, the thyroid gland was surgically exposed. The right lobe and the isthmus appeared quite normal; the left lobe was found to be diffusely enlarged, of a yellowish-rosy discoloration and firm to touch. A tissue specimen was taken from the left lobe.

Biopsy (Dr. L. Kende): side by side with the picture of a chronic thyroiditis (i.e., degenerative changes of the follicular epithelium, formation of giant cells and of intrafollicular pseudotubercles), true tuberculous granulation tissue was seen (epithelioid cells, giant cells of the Langhan's type and central necrosis). No tubercle bacilli were found.

The patient was treated with dihydrostreptomycin (one gram twice a week), nicotibine (300 mg daily) and PAS (12 grams daily). The operative wound healed in a week's time. Two weeks after the operation the patient left the hospital in a good condition; the left thyroid lobe was hardly palpable and not tender.

Follow up: three months after operation the patient's general condition was good. She received regularly streptomycin, nicotibine and PAS (in doses indicated above). The thyroid gland was of an uniformly soft consistency and not tender. The second tracer test showed that 36 per cent of the radioactive iodine were concentrated in the thyroid gland after two hours and 60 per cent after 24 hours. This, according to our laboratory standards, indicated an uptake higher than normal. The profile showed a normal uptake throughout the whole region of the thyroid gland with some increase over the right lobe.

These results suggested a functional recovery of the left thyroid lobe.

COMMENT

The appearance of the clinical picture of thyroiditis characterized by a prolonged course without acute, severe inflammatory signs in a patient with a history of tuberculosis elsewhere in the body, should arouse the suspicion of a specific process in the thyroid gland. If a suitable course of specific treatment fails, surgical exploration of the thyroid may be done in order to establish the definite diagnosis and to exclude the possibility of a malignant neoplasm, which may masquerade as a chronic thyroiditis. Bacteriological and virological culture studies on biopsy material are indicated. Unfortunately, in the present case this was not done.

SUMMARY

A case of caseous tuberculous thyroiditis is described. The etiology of the illness was suspected on clinical grounds and supported by histological examination. The satisfactory result obtained with the antituberculous therapy (dihydrostreptomycine, nicotibine and PAS) gave additional evidence to the diagnosis.

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XIX

THE CREATION OF A MESIAL CRUS "ARM" TO PREVENT THE NASAL TIP FROM DROPPING

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The rhinoplastic surgeon is confronted with the ever-perplexing problem: the means of preventing the nasal tip from dropping. There are almost as many reasons given for this occurrence as there are differences in technique. Some explanations advanced to avoid dropping of the tip are: positioning a button over the reshaped nasal tip, a batten or strut placed in the columella, making an incomplete transfixion of the nose, removing excessive mucous membrane from the caudal end of the septum to avoid scar tissue contraction, the preservation of the vestibular skin of the lower laterals, making certain that the lower lateral cartilages are swung over the upper lateral cartilages (the idea is a good one, yet it cannot be done if incised at the angle through the vestibular skin), that the caudal end of the upper laterals are not cut too short, suturing the columella at a higher level on the septum, short columella, tension of tip muscles and many others.

Most everyone who delivers the lower lateral cartilages has experienced that after the nasal tip has been properly remodeled and replaced it has flattened out to a greater or lesser degree, necssitating building it up.

The anatomical relationship between the upper and lower lateral cartilages is well understood by everyone. Yet, this anatomical relationship is deliberately destroyed without realizing it is being done. If one will always remember that the upper lateral cartilages support and maintain the position of the nasal tip cartilages by the fibrous tissue between these, the problem of a dropping tip is solved.

In a typical type nasal plastic case, nasal parts can be removed or otherwise shifted into position to produce the desired result without

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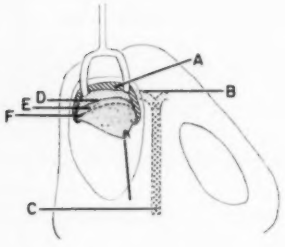

Fig. 1.—Lower lateral delivered through rim incision. A. Vestibular skin of rim incision. B. Caudal edge of upper lateral. C. Caudal end of septum. D. Cephalic border lower lateral cartilage. E. Exposed edge of undersurface of lower lateral cartilage. F. Cephalic edge of vestibular skin on undersurface of lower lateral cartilage.

grafts, providing no defect has been created by the surgery. The placing of grafts in the tip of the nose, autogenous or otherwise, to create an effect signifies some shortcoming in the technique. Converse[1] mentions various techniques for the correction of problems of the nasal tip.

The technique which I shall describe takes into consideration the preservation of the anatomy of the nose that is present before surgery and maintaining that anatomical relationship after its completion. This basic principle has been successfully carried out for the correction of the bony vault, but the difficulty seems to be with the surgery of the lower laterals.

There are two important factors to be considered, to prevent the nasal tip from dropping.

1. Removing the excess vestibular skin from the cephalic border of the lower lateral cartilage.

When the lower lateral cartilage is delivered, a small portion of the cephalic border is always found exposed, devoid of any vestibular skin. Therefore, whenever the cephalic border of the lower lateral cartilage is reduced or otherwise changed, any excess vestibular skin which is present after the modeling, must be removed, uncovering a similar portion of the cartilage that was originally exposed (Fig. 1).

The lower lateral when replaced should heal to the upper lateral in the same position it was before being separated at the lineum vestib-

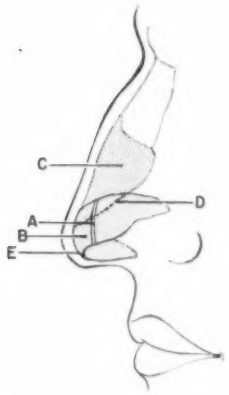

Fig. 2.—Normal nasal anatomy showing mesial crus arm. A. Incision through lateral crus of lower lateral cartilage. B. Mesial crus arm resting on upper lateral cartilage. C. Upper lateral cartilage. D. Caudal border upper lateral cartilage. E. Angle lower lateral cartilage.

ulae. When raw surface faces raw surface proper healing takes place but skin will not heal to a raw surface. The delivered lower lateral cartilage has a raw area on both its superior and inferior surface. Its superior surface, having a greater raw area, will when replaced unite and heal together with the overlying subcutaneous tissue of the skin, while the raw area of the inferior surface, although smaller in area, will firmly unite and heal with the existing raw area of the caudal edge of the superior surface of the upper lateral cartilage. In this manner a natural two-point attachment with firm fibrous union is established between the lower and upper lateral cartilages, holding the nasal tip in its new position and preventing it from dropping. If on the other hand the vestibular skin is intentionally allowed to remain, as is so frequently done, only the superior raw area of the lower lateral cartilage will unite and heal together with the overlying raw area of the subcutaneous tissue, but the inferior surface of the lower lateral with the excess vestibular skin present will not unite and heal with the raw area of the caudal end of the upper lateral; instead the skin surface will merely temporarily rest on the edge of the upper lateral. Thus we have a raw area healing superiorly with the overlying subcutaneous tissue, with no healing of the inferior or vestibular skin surface to the caudal edge of the upper lateral cartilage. This is a weak union with the upper lateral, because there is only one point of attachment above and none below. There should be two points of attachment.

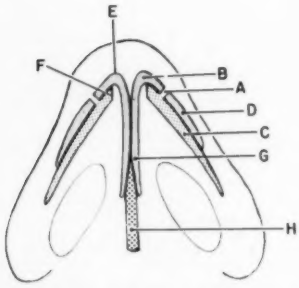

Fig. 3.—Cross section of nasal tip at level of caudal end of upper lateral showing position of mesial crus arm over the upper lateral cartilage. A. Incision through lateral crus. B. Mesial crus arm resting on upper lateral cartilage. C. Upper lateral cartilage. D. Lateral crus. E. Angle lower lateral cartilage. F. Incised upper lateral from septum. G. Mesial crus. H. Nasal septum.

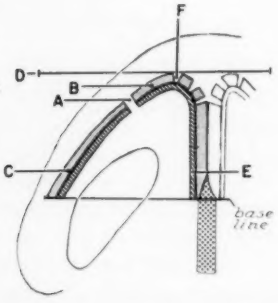

Fig. 4.—Cross section of delivered lower lateral cartilage, showing cartilage removed from mesial crus arm to reduce projection. A. Incision through lateral crus and vestibular skin. B. Mesial crus arm. C. Lateral crus. D. Level of tip before reduction. E. Vestibular skin. F. Sections of cartilage removed from angle and mesial crus, leaving the vestibular skin.

The vestibular skin surface will gradually and eventually slip off the upper lateral, with scar tissue contraction from above, pulling down the overlying subcutaneous tissue, causing the tip to drop. Where the vestibular skin is kept intact, it is necessary that the inferior surface of the cephalic border of the lower lateral cartilage be devoid of vestibular skin to get union at this point. Saving the vestibular skin of the cephalic border of the lower lateral cartilage is anatomically and surgically incorrect.

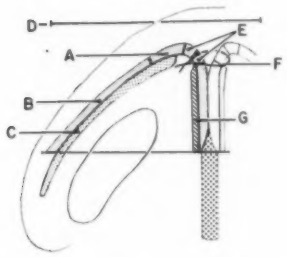

Fig. 5.—Cross section of repositioned lower lateral cartilage. Projection reduced showing position of mesial crus arm. A. Mesial crus arm resting on upper lateral cartilage. B. Lateral crus. C. Caudal end of upper lateral cartilage. D. Level of tip before reduction. E. Sections of cartilage brought together by contraction of vestibular skin. F. Suture through upper part of both mesial crura. G. Vestibular skin.

2. Replacing the mesial crus, to override the upper lateral lateral cartilage, where it belongs, by the creation of a mesial crus "arm."

Understanding the first principle, the next important step to follow is to make certain that the continuity of the mesial crus with the lateral crus is maintained in its entirety, to permit them to override the caudal edge of the upper laterals where it normally belongs. This anatomical continuity gives support to the nasal tip (Fig. 2).

It has been routine, after the delivery of the lower lateral cartilages, to meticulously place a hook at the angle and then cut through the cartilage and vestibular skin at this point. This is referred to as cutting through the angle.

In those cases where the lower laterals are cut through the cartilage and vestibular skin at the angle, the mesial crura will immediately lose the continuity of the lateral crus making it free, (it "floats") and impossible to again override the caudal edge of the upper lateral. This procedure causes the tip to drop at the operation and postoperatively. To overcome this error in technique, the lower laterals should never be cut at the angle; instead, the lower lateral should always be cut lateral to the angle, thus creating an "arm" of the mesial crus, long enough to enable this portion of the lower lateral to rest on the upper lateral, the position it normally holds (Fig. 3). Modeling of the tip can be carried out as usual.

The mesial crus arm must rest on the upper laterals after the projection of the tip is lowered or increased as follows:

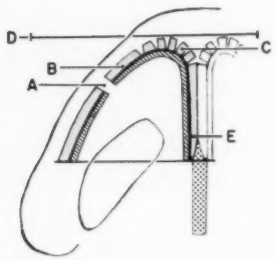

Fig. 6.—Cross section of delivered lower lateral cartilage showing cross-hatching of mesial crus arm to increase the projection. A. Incision through lateral crus. B. Mesial crus arm. C. Cross-hatching of cartilage above and below angle to vestibular skin. D. Level of tip before projection. E. Vestibular skin.

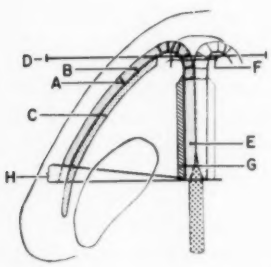

Fig. 7.—Cross section of repositioned lower lateral cartilage. Projection increased showing position of mesial crus arm. A. Incision through lateral crus. B. Mesial crus arm resting on upper lateral cartilage. C. Upper lateral cartilage. D. Level of tip before projection. E. Mesial crus. F. Suture through upper part of both mesial crura. G. Vestibular skin. H. Amount of shortening of lateral crus to effect increased projection.

The projection can be lowered by removing small pieces of cartilage from the mesial crus arm and angle, leaving vestibular skin between the remaining pieces of cartilage (Fig. 4). When the vestibular skin of the mesial crura arm on both sides contracts, it will reduce the projection, the degree depending upon the amount of cartilage removed. A suture can then be placed through both mesial crura to secure proper apposition at the desired level (Fig. 5). The mesial crus arm, when replaced, will rest on the upper lateral cartilage, and become anchored there.

Any interposing tissue between the mesial crura can be removed as required and the mesial crura sutured together to eliminate a cleft of the tip. Any portion of the lateral crura requiring modeling can also be carried out as usual.

To increase the projection, the angle and mesial crus arm are cross-hatched to the vestibular skin, without removing cartilage. In this instance there should be more of the lateral crus present beyond the angle, by incising the lateral crus further from the angle laterally, to leave a sufficiently long mesial crus arm for it to rest on the upper lateral when replaced. It is then turned inward and upward and sutured together at a point lateral to the anatomical angle (Figs. 6 and 7).

If the upper lateral is short, the mesial crus arm must be made long enough to permit it to rest on the caudal end of the short upper lateral cartilage.

A small vaseline gauze packing is placed into each nostril.

The usual adhesive tip dressing will retain the parts in their new position.

SUMMARY

1. To obtain the desired result one wishes to attain, the physical anatomy of the lower lateral cartilage must not be destroyed.

2. The mesial crura, when cut at the angle, lose the support of the upper lateral cartilage and, therefore, drop to a lower level after surgery and drop still more after healing. They should never be cut at the angle.

3. By including a portion of the lateral crus when incising the lower lateral cartilage, an arm is created lateral to the angle, which rests on the caudal end of the upper lateral cartilage and becomes fixed to it. The created arm of the mesial crus retains the normal relationship with the lateral crus of the lower lateral cartilage (Fig. 6).

4. It is not necessary to replace tissue that has been removed or other grafts, to build up the tip.

5. Increased projection is obtained at the expense of the lateral crus, in order to get a mesial crus arm long enough to place over the upper lateral.

6. The reduced projection requires less of the lateral crus to obtain a mesial crus arm to set on the upper lateral. The sections of cartilage removed from the mesial crura permit the vestibular skin, from where the cartilage was removed, to contract, thereby reducing the projection.

7. The cephalic border of the lower lateral cartilage must be devoid of vestibular skin to get a two-point attachment for firm union.

8. The nasal tip can be maintained at any desired level of projection without it subsequently dropping, with the technique described, by the creation of a mesial crus arm devoid of vestibular skin along its cephalic border.

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XX

THE PRESSURE TEST IN MUTATIONAL VOICE DISTURBANCES

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Mutation, the change of voice, is the most important landmark in the vocal history of an individual. During the stormy and emotionally charged period of puberty the voice of the child matures and is transformed into the fully developed voice of the adult.

The phenomenon of mutation has attracted the attention of scientific observers since antiquity. The voluminous literature (more than 300 references) can be found in the comprehensive review by Weiss.[1] In the vast majority of instances the transformation is a gradual process that occurs without audible "breaks" at the age of 13 to 15 in boys and 12 to 14 in girls. The cracking "Henry Aldrich" voice of the adolescent is the exception rather than the rule. The change of voice begins earlier than has been generally assumed. Fairbanks[2] has demonstrated minor breaks as a frequent occurrence in seven and eight year old boys and girls. The noticeable "change" in the voice is merely the acceleration of a rather slow process that quickens during puberty and continues, at a much slower rate, for a few years afterwards. Many deviations from the normal process of mutation have been observed in both sexes. The voice of boys drops during mutation by an average of an octave but all kinds of abnormalities occur. Dysfunction of the sex glands may delay or inhibit the change of voice together with the other secondary sex characteristics. Or, in spite of otherwise normal pubertal development and normal growth of the larynx, the voice may not descend at all (persistent falsetto voice, better called postmutational falsetto voice) or drop by a few notes (incomplete mutation). Or it may descend below the "normal" level (mutational basso). Similar abnormalities have been observed in female voices. Incomplete mutation has been found in female voices, too, although it is less conspicuous

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than its male counterpart. And the descent of the female voice to an abnormally deep level has been described as perverse mutation.

If we exclude the relatively rare hormonal insufficiencies, there is general agreement on the psychogenic basis of the abnormalities of mutation. The earlier authors, such as Gutzmann,[3,4] Flatau[5] and Makuen,[6] who described the persistent falsetto voice, have already stressed this connection.

With growing interest in the pathology of mutation more attention has been paid to the emotional dynamics of incomplete mutation. While the postmutational falsetto voice is rather uncommon, the incomplete mutation is a very frequent occurrence. Weiss[1] has stressed the importance of this incomplete maturation of the voice as the basis of countless functional voice disorders that develop in adult life. The adult with a fully grown larynx who continues to speak with an abnormally high voice is liable to develop more or less severe voice disturbances, particularly if he uses his voice extensively or for highly specialized tasks. If he becomes a singer he will join the ranks of the pseudotenors who, sooner or later, will most likely experience vocal difficulties.

The adult with postmutational voice disorder carries the emotional conflicts of his puberty into his adult life in the form of an abnormal vocal pattern. Unwillingness to face the realities and responsibilities of adult life, mother-fixation, hatred of the father, sexual ambivalence rank high among the many conflict situations that may leave their imprint on the voice of the adolescent. The adult may still carry the unconscious burden of such conflict situations or he may have outgrown them. But his voice still mirrors the emotional upheavals of his puberty.

Modern psychiatry has taught us to understand the impact of the conflicts of childhood and adolescence on the emotional problems of the adult. This applies also to the disturbances of the voice. In many of these cases the pattern of adult vocal behavior has become fixed during the pubertal change of the voice; the mechanical factors of abuse or misuse of the voice should be evaluated against the background of earlier disruption of vocal function during mutation. For this reason the abnormalities of mutation assume an important role in the diagnosis of vocal disorders of the adult.

It has been known for a long time that backward pressure on the thyroid cartilage during phonation lowers the pitch. It counter-

acts the tension of the cricothyroid muscle. For this reason the use of pressure against the thyroid cartilage has already been recommended by Gutzmann[4] and Makụen[6] to obtain a deeper voice at the start of the treatment of the postmutational falsetto voice. In addition, Tarneaud[7] has advocated a downward pressure on the larynx to counteract the excessive rise of the larynx during phonation in falsetto voices. However it should be mentioned that most authors agree that such a mechanical lowering of the voice is, at best, only a technical short-cut to speed up the initial phase of the treatment. As Froeschels[8] has pointed out it may even lead to the establishment of a hypofunctional pattern of voice production. Moses[9] has underlined the need for an attack on the underlying psychological situation to effect a permanent cure.

Gutzmann[10] was the first to use pressure on the thyroid cartilage as a diagnostic test. He observed the following phenomenon in phonasthenic voices: if one asks a person with a normal voice to produce a singing tone while exerting pressure on the thyroid cartilage and then releases the pressure, the voice will go up for only a fraction of a second, and the original pitch will be resumed immediately. In phonasthenic voices the original pitch will be resumed only after a longer delay or, in severely damaged voices, not at all.

The observation reported in this paper is a kind of Gutzmann test in reverse. It was first made in 1954 on a patient with an excessive and very obstinate postmutational falsetto voice who presented many features of a prepsychotic (schizophrenic) condition. After other means of inducing a deeper pitch (phonation of low vowels, chewing exercises) produced not result, pressure on the thyroid cartilage was tried. To our surprise the voice of the patient, instead of going down, went up sharply for a few seconds and then resumed the original pitch. The unconscious urge to speak in an abnormally high voice was apparently so strong that it overcame even the mechanical lowering of vocal cord tension by the pressure on the thyroid cartilage. At a demonstration of the patient by the author[11] that took place after a number of therapeutic sessions the patient still presented this paradoxical voice pattern.

In a personal communication van den Berg (Univ. of Groningen, Netherlands) suggested a possible explanation of the pressure test in terms of muscular physiology: If the balance between the opposing forces of longitudinal and lateral cord pressure is disturbed in favor of the latter the decrease of longitudinal pressure (which lowers the

voice) by the test would be overcompensated by excessive lateral pressure. If this pressure is too high, particularly in the neighborhood of the arytenoids, the vibrating part of the cords would be shortened with resulting raise in pitch.

Since then the pressure test has been used in the first examination of patients with all kinds of functional voice disturbances. In all patients with voice disorders of nonmutational origin the test was negative: on application of pressure during phonation the voice followed the normal pattern of initial lowering of the pitch. Of 24 male patients with postmutational voice disturbances (of the mutational falsetto and the incomplete mutation type) 20 gave a positive response: the paradoxical initial rise of pitch on application of pressure.

Of these 20 patients three were classified as having postmutational falsetto voices, the rest as incomplete mutations. Five of the patients with incomplete mutation were young singers who could be classified as pseudo-tenors, i.e., singers with the physical and vocal characteristics of genuine baritones who had been encouraged by their teachers to sing as tenors and who had experienced vocal difficulties after a few years of singing.

The last nine patients of the group which gave a positive pressure test were then re-tested with Lombard's test (temporary deafening of both ears by masking noise). Six of them maintained the pitch rise on pressure even under these conditions.

Froeschels who witnessed the first observation of the pressure test and stimulated its follow-up has used the test on his own patients. He found it positive in all cases with definite mutational voice disturbance of the incomplete mutation type.

The pitch rise varied between a halftone and two tones. After the first therapeutic sessions the test became negative in all patients with incomplete mutation. In other words, with the restoration of a more normal voice the response to pressure during phonation changed into the normal pattern of lowering of pitch under the influence of pressure. As was to be expected, the postmutational falsetto voices maintained the positive test for a longer time.

The four patients with a negative response to the test during the first examination were all people in whom the mutational factor was of only minor importance, with one interesting exception. This was

a well-known counter-tenor who sings, with the long vocal cords of a baritone, in an excessively high voice which reaches the e^2 without difficulty. This artist who sings with an excellent technique and with great clarity and accuracy, but speaks with a voice that betrays the mutational character in its frequent mixture of high and deep sounds, has, so far, experienced no vocal difficulties. He was tested during an investigation of his unusually interesting voice and kept the pitch of any chosen sound even under strong pressure. Apparently, a singer with such a highly trained singing voice is too much on guard against any interference with the maintenance of pitch to be influenced by the pressure test.

The observation that is reported in this paper may be evaluated as a new proof of the psychological character of the postmutational voice disturbances. Moses[12] has recently introduced the concept of the "slip of the larynx" and speaks in this connection of the "breakthrough of the original impulse." It seems that, in the postmutational voice patient, the unconscious urge to use a higher voice is so strong that it overcomes even opposing mechanical forces with a breakthrough into the higher pitch. This is not astonishing if one keeps in mind that the patient who speaks with a voice that is higher than warranted by his larynx and his vocal cords frequently develops all kinds of vocal difficulties, faces the ridicule of his environment, and even experiences unpleasant physical sensations, ranging from irritation to real pain. The resistance that many of these patients put up against the normalization of their voices by vocal therapy proves the power of the unconsciously motivated urge to maintain the high voice level at all costs. The positive pressure test is only a new confirmation of the psychodynamics of the postmutational abnormalities of the voice.

SUMMARY

Pressure on the thyroid cartilage during phonation produces, in normal voices, an initial lowering of the voice. In patients with postmutational voice disturbances of the mutational falsetto and of the incomplete mutation type a paradoxic pattern of initial pitch rise on pressure was observed. The observation is interpreted as a confirmation of the psychogenic character of these voice disturbances. The test, aside from presenting an interesting phoniatric symptom, is helpful in diagnosing the mutational basis of voice disturbances.

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XXI

A REAPPRAISAL OF THE CLINICAL AND MORPHOLOGICAL CLASSIFICATION OF THE TYMPANIC SPACES AND THE EUSTACHIAN TUBE

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It is proposed here to present evidence to the effect that the current classification of the tympanic spaces and the eustachian tube is basically wrong and untenable. In conclusion another alternative classification will be suggested and the clinical importance of this change will be emphasized.

A cursory historical retrospection seems first of all necessary to get at the roots of: 1) how the current classification came into being and 2) how this classification, wrong though it was, could survive unchallenged till today.

The tympanic cavity proper has been described, as we all know, by Fallopius. He compared its shape to the military drum. Only several years later G. Eustachio published his classical description of a canal connecting the rhinopharynx with the tympanic cavity (Venice 1563). It should be pointed out that he described and knew of the membrano-cartilaginous canal exclusively.

Both these descriptions fell practically into oblivion until, after about 150 years, the famous scientist and anatomist, A. M. Valsalva, unearthed the forgotten items in 1717 and, in his "Tractatus De Aure Humana," presented an entirely new concept of the tube. Valsalva discovered a new anatomical entity known to neither Fallopius nor Eustachius and connecting both of the previously described items. He called this connecting section the "bony tube" and presented the merger of this section with the cartilaginous tube as the "eustachian tube."

Now this arbitrary merger of his own making is, as we will see, a blunder. Valsalva was a great anatomist, but let me recall that

investigation into the province in question was and has remained technically difficult and that anatomical dissections were in Valsalva's time charged with almost insurmountable handicaps. We should not overlook either that the main viewpoint of Valsalva was anatomical. Microscopy and embryology had not yet been developed. Clinical otology did not exist.

It is nevertheless surprising that in the course of more than two centuries no criticism about Valsalva's merger has been attempted. For instance, Arione, who based his observations on not less than 150 temporal bones examined, as late as 1923 entirely overlooked the problem of classification.

Scrupulous authors, like Ruedinger who published his extensive papers between 1867 and 1872, passed lightly over the striking discrepancy between the microscopic picture and the bony tube.

In order to stress my point, let me make here another remark. Considering modern otology, one is struck by the contrast between the brilliant development and abundant output of the literature about the tympanic spaces and the scarcity and relative insignificance of the publications about the tube. The explanation of this contrast is, in my opinion, to be sought in the centuries-old prestige of the famous Valsalva.

I have sectioned systematically a hundred temporal bones of human fetuses and adults, as well as several of the dog, each one in continuous series of slides, macro- and microscopically, and here are my conclusions concerning classification.

Let me start with two basic statements:

1) The fundamental boundary between two definitely heterogeneous provinces is the isthmus, as long, of course, as pathological processes do not interfere.

2) There exists no such dividing boundary, as the "tympanic orifice of the tube," the latter being only a natural, in most cases vastly open, communication between essentially homogeneous sections of an over-all pneumatic cavity, carved out within the temporal bone: the middle ear system. The traditional concept of an "ostium tympanicum tubae" as dividing point is wrong and misleading and should therefore be abandoned, and here are the arguments in support of my contention:

Morphologically, the lumen of the membranous tube is, in repose, even in its fornix a closed capillary slit designed to open periodically, by muscular action. The bony tube, on the other hand, is a rigid, permanently open, bone-walled pneumatic space, exactly like the tympanic cavity and the cell-system of the temporal bone.

Histologically, forty thousand serial slides comprising the area from the pharyngeal orifice of the tube up to the mastoid antrum, among them five thousand stained by various methods and examined microscopically, proved beyond doubt that the tube, medially from the isthmus, is supplied with a true mucous membrane with columnar ciliated epithelium, a submucosa, muco-serous glands and rich glandular tissue, such lining being strictly identical with that of the upper respiratory tract, whereas the lining of the tube, distal to the isthmus (the bony tube) is identical, or almost identical, with that of the tympanic cavity proper, both of them being, in fact, not mucous membranes at all, as long as physiological conditions prevail, with a single layer of flattened, or at the most cuboidal, cells adherent directly to the endosteum and with no trace of cilia, with no submucosa, no glands or adenoid tissue. It is obvious that this picture constitutes the sharpest possible contrast with the mucosa of the membranous tube. Lest the above statements seem superfluous, I wish to submit a quotation from Gray's Handbook of Anatomy, 28th edition, which is a misstatement and evidence of the confusion prevailing sometimes in this domain. It reads, "ciliated epithelium lines the respiratory tract . . . the tympanic cavity . . . and the pharyngotympanic tube."

Embryologically, a fundamental difference prevails between the two sectors of the tube. Time does not permit me to dwell on some of these findings. It may suffice to point out that the bony tube is entirely integrated into the spectacular process of pneumatization of the temporal bone, a process that is completely different from that in the membranous tube.

Remarkable from the viewpoint of phylogenesis is a striking analogy prevailing between the tracheobronchial rings and the muscular breathing mechanism of the respiratory tract on the one hand, and the semicylindric cartilage and the periodical air-intake, called ventilation, of the membranous tube on the other, as evidence of the common branchial origin of both structures.

The bony tube, on the other hand, is an entirely different device committed in its development to the pneumatization process of the temporal bone.

Even more important are the clinical findings which were in fact the starting point of my investigation.

From the clinical point of view a sound classification, although seemingly a formal affair, is crucial. Wherever pathological or clinical classification of issues related to the tube is involved, there confusion or evasiveness often prevails. This occurs, I suggest, because of lack of a clear-cut distinction between conditions located primarily in the membranous tube, and those peculiar to the altogether different nosology of the pneumatic system of the temporal bone, including the bony tube.

It is not proposed to set forth here all the implications. Some examples may be mentioned: 1) Frenckner, Ramadier, Almour-Kopetzky, Lempert, authors who devised surgical approaches to petrositis, will, I am sure, admit that the so-called peritubal tip-cells are an integral part of the pneumatic system of the temporal bone; 2) Zöllner, Wullstein, and Pietrantoni will certainly not deny that the sometimes overlooked bony tube represents an essential air reserve space in the functional middle ear plastic surgery; and 3) I suggest that in the course of a "secretory otitis media" it is useful to realize that the isthmus, and not the so-called "tympanic orifice of the tube," is clinically the dividing point between the etiologic factor of an obstructed membranous tube and the pathologic effect of transudation taking place not only within the tympanic space proper but also in the funnel-like cavity of the bony tube.

The last evidence in support of my contention is that of comparative anatomy: among vertebrates in general and mammals in particular we nowhere encounter a structure to be likened to the bony tube in man, the tympanic cavity passing, wherever it exists, directly into the membranous tube. So, we may ask, what is the bony tube in man, after all? It is, in my opinion, an accidental deformation of the medial-anterior segment of the tympanic space proper, brought about originally by the intrusion of a highly developed internal carotid artery, such expansion being set in motion by the exorbitant demands of blood supply to a uniquely developed forebrain in man.

CONCLUSIONS

In accordance with what has been said, and recalling the previously mentioned, basic statement, I suggest a reclassification of the tympanic spaces and the eustachian tube along the following lines:

1) The established merger of the cartilaginous and bony tube shall be repudiated.

2) By the term "eustachian tube" we shall henceforth signify its section located anteromedially from the isthmus exclusively, thus going back roughly to the authentic concept of Eustachius.

3) An unfounded, if time-honored, partnership being dissolved, the bony tube located distally to the isthmus shall be merged, morphologically as well as clinically, with the whole pneumatic area of the temporal bone.

4) This whole pneumatic system we may, as a matter of expediency, divide into a central, axial area, which we may call "Pantympanum," and a peripheral one: the cell-system of the temporal bone (into which the cells of the so-called bony tube shall, of course, be included).

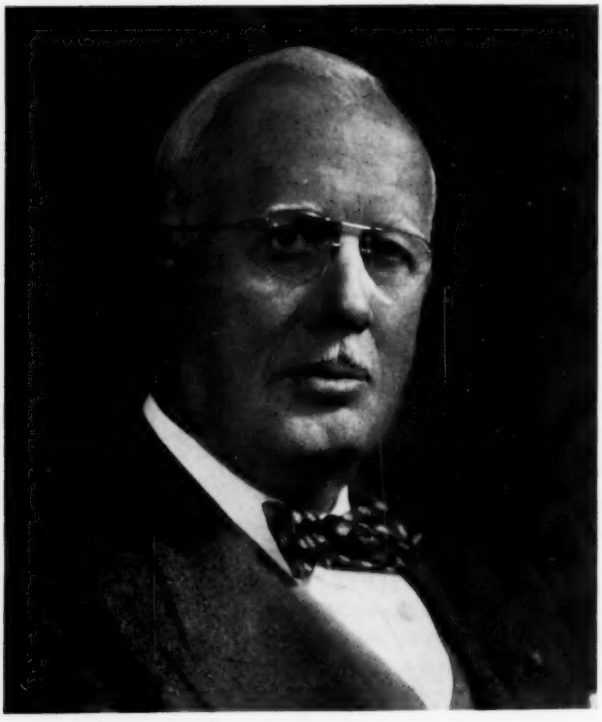
5) Within the axial area of this system we shall envisage a space placed centrally and corresponding roughly to the "tympanic cavity" of Fallopius, and call it "Mesotympanum." Around the mesotympanum we shall group its three extensions: an upper, a lower and an antero-medial one. The denominations of the first two are generally accepted as epitympanum (attic) and hypotympanum. The third one (the former bony tube) we might name "Protympanum."

New word-coinings are, of course, disputable.

The implications, morphologically as well as clinically, involved in this reclassification seem to me manifold and, partly, far-reaching. It is not proposed to discuss them here.

In winding up this analysis I am aware of the difficulties standing in the way of dislodging a universally established terminology and classification.

5 Rosenbaum Street



HENRY BOYLAN ORTON

HENRY BOYLAN ORTON

1886 - 1957

Henry Boylan Orton convinced his physicians that an injury from a needle-prick suffered at an operation one hundred twenty days before the onset of his last illness was the cause of a virus hepatitis. Seven weeks from the onset of this illness, complications partly due to residual kidney disease following transfusions at the time of his severe mastoiditis in the late nineteen twenties caused his death on September 1, 1957.

On February 6, 1886, he was born in Newark, New Jersey. His pride of ancestry is evidenced by his membership in the Founders and Patriots of America, the Society of Colonial Wars in the State of New York, the Sons of the American Revolution, and the New Jersey Historical Society. His English ancestors were prominent in colonial affairs and engaged in the Colonial Wars and the American Revolution. This pride extended to his personal appearance and conduct, which was meticulous and exemplary.

Doctor Orton received his early training in Newark Schools and obtained his medical Degree from Jefferson Medical College in Philadelphia in 1908. He began his career as a general practitioner in East Orange, New Jersey, but soon interested himself in diseases of the ear, nose and throat. His interest was furthered by an assignment in this field as Captain in the American Expeditionary Forces in France during World War I, where he operated upon a large number of mastoids.

After his tour of duty he transferred his practice to Newark, and developed skill in bronchoscopy after courses with Chevalier Jackson in Philadelphia. Prolonged visits to Paris, London, and other clinics in this country and abroad furthered his excellence in laryngology. His appointment in bronchoscopy to the Newark Eye and Ear Infirmary and Newark Presbyterian Hospital enabled him to pursue his

clinical studies where his energy and devotion brought international recognition.

He was author of many articles and is especially known for his promotion of the "wide field" laryngectomy for carcinoma of the larynx. Dr. Orton gave unstintingly of himself in the care of his patients and demanded high standards of service from physicians and nurses with whom he was associated.

Doctor Orton soon became aware of the problem of rehabilitation of laryngectomized patients and instruction in the use of pharyngeal (esophageal) voice was given in classes at his office as early as 1925. Classes became so large that space was set aside for their use at Newark Presbyterian Hospital in 1947. Doctor Orton was inordinately proud of this Speech Clinic which has registered over one thousand laryngectomies.

Following the example of Mosher and Jackson, he found painting a relaxing hobby. Professional instruction and his attention to detail plus natural ability produced prize awards in several exhibits.

In his long professional career he brought service with distinction to New Jersey where he was consultant to many hospitals. In Newark he served as Attending Physician on the staff of the Presbyterian Hospital, the Newark Eye and Ear Infirmary, the Newark City Hospital, and St. Michael's Hospital. He was Clinical Professor of Laryngeal Surgery at New York Polyclinic Medical School and Hospital.

He took a leading role in national and international affairs in his field, and at one time served as President of the American Laryngological Association and of the American Broncho-Esophagological Association. He was also a member of the American Board of Otolaryngology, and organizer and first President of the New Jersey Society of Ophthalmology and Otolaryngology.

Among honors he received are the Casselberry and the Newcomb Awards of the American Laryngological Association and the Edward J. Ill Award of the Academy of Medicine of New Jersey. He also

held the honor key of the American Academy of Ophthalmology and Otolaryngology.

A bronze plaque commemorating the work with laryngectomies, to be unveiled in February, bears the following inscription:

A GRATEFUL TRIBUTE

BY THE BOARD OF TRUSTEES AND
THE MEDICAL STAFF OF THIS HOSPITAL

IN MEMORY OF
HENRY BOYLAN ORTON, M.D., F.A.C.S.
FEBRUARY 6, 1886 - SEPTEMBER 1, 1957

WHOSE SCIENTIFIC ATTAINMENTS AND ABIDING
INTEREST IN THE WELFARE OF HIS PATIENTS
CONTRIBUTED SO MUCH TO THE REHABILITATION
OF PERSONS WHO HAD UNDERGONE LARYNGECTOMY

E. P. C.

Society Proceedings

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Meeting of Monday, February 4, 1957

THE PRESIDENT, DR. RAYMOND W. KERWIN, IN THE CHAIR

Ossification of the Labyrinth in a Case of Glomus Jugulare Tumor

T. H. BAST, M.D.

(Abstract)

During the past eight years many cases of glomus jugulare tumor have been recorded, but none in which there was also observed labyrinthitis ossificans. This latter condition was first described by Politzer in 1887 in a 13 year old boy who at the age of two and a half years was severely ill with convulsions. Numerous similar cases were reported by others around the turn of the century and always with the history of severe infections of the cerebrospinal meningeal type during the early years of life. In a case reported here such ossification apparently did not occur until late in life. The patient died suddenly July 7, 1955, at the age of 60 years, two days after a second mastoidectomy.

The main entries on the patient's medical record show tonsillectomy, 1929, uterine fibroids with hysterectomy in 1930, and stilbestrol medication since the latter date. Her ears became stopped up in 1932. Hearing loss and pounding tinnitus in left ear gradually increased between 1937 and 1945. In 1947 a husky voice was noted. The tongue became thicker, there was difficulty in swallowing, and serous discharge from the left ear. The patient veered to the left in walking. Polyp was removed from left ear in 1949. She entered Wisconsin General Hospital for the first time in February, 1948, and was transferred to the Otolaryngology Department in May, 1948, where the initial impression was middle ear disease with polyp. X-ray

showed active infection in the left mastoid. Basal skull films were negative. A very vascular polyp was removed from the left ear. Radioactive phosphorus (P_{32}) was given with resulting decrease in size of mass for a short time, but there was no change in the mastoid. Radical mastoidectomy was done July 15, 1949, and as much of the friable vascular tumor mass removed as possible. It had invaded the floor of the middle ear from the jugular bulb region. The condition remained quiescent until December, 1954, when a large swelling appeared on the left side of the neck together with reappearance of the tumor of the left middle ear. There was neurological evidence of involvement of V to XII cranial nerves. On January 3, 1955, a neck dissection was done with partial removal of a tumor mass involving the left jugular vein. The diagnosis was glomus jugulare tumor. X-ray therapy consisting of 3000 r was given in twelve treatments. The tumor in the left ear continued to enlarge and vestibular involvement became more marked. A second and final mastoid exploratory operation was performed July 5, 1955, and was followed by death two days later.

At autopsy the left petrous bone was removed, prepared and sectioned in series. The tumor had invaded all petrous air cells and even the bone marrow of the petrous apex. The interesting fact, however, was that all portals of entry to the labyrinth, viz., secondary tympanic membrane, aquaductus cochleae, aquaductus vestibuli and internal acoustic meatus, were resistant to the tumor and no tumor tissue was found anywhere in the labyrinth. Instead, the entire labyrinth was filled with new bone with the exception of a small part of the vestibule. This labyrinthine ossification apparently developed independently from the glomus tumor.

The cause of this ossification is not known. The sections did show that the stapes was dislocated along its anterior margin, probably due to middle ear packing following the tumor removal. A dense, well-developed but vascular connective tissue filled the gap between the oval window and the displaced part of the stapes. It is possible that an infection could have entered the labyrinth via this ruptured annular ligament and that this infection may have initiated the ossification. The only objection to this etiology is the fact that the new bone which fills the labyrinth did not invade the area where the stapes ligament was ruptured. Thus, the origin of the ossification remains unknown. In the future it will be of interest if labyrinthitis ossificans will be noted in cases of otitis media following dislocation of the stapes in attempted stapes mobilization.

DISCUSSION

DR. JOHN R. LINDSAY: This case which Dr. Bast has presented reminds me of a case which we treated before the days of penicillin which had a similar, long history of previous operations, a facial paralysis of several years' duration and a dead ear. We thought we were dealing only with a suppurative problem, but on operation we found we were dealing not only with granulation but with tumor tissue. That case came to a fatal termination from meningitis. The histological study of the ears showed labyrinthitis ossificans such as was found in Dr. Bast's case, but also a subluxation of the stapes, and I feel that it is likely that the same thing occurred at the previous operation in Dr. Bast's case. In controlling the severe bleeding with packing, the stapes may easily be subluxated.

The labyrinthitis was the result of infection of the labyrinth rather than due to tumor.

We have examined several cases of tumor of the temporal bone, where the tumor literally surrounded the capsule, but it is surprising how resistant the endosteum is to invasion by the tumor. It seems that this dense, almost avascular bone, can be extensively eroded before the tumor is able to invade the labyrinth. Then, again, we have some cases, only a few, where the labyrinthitis ossificans came about as a result of invasion by infection in the petrous pyramid. Many of the cases of labyrinthitis ossificans reported in the literature were due to meningococcic meningitis from which the patient had recovered but had developed a dead labyrinth. I am of the opinion that ossification of the labyrinth in this case came about as a result of tympanogenic labyrinthitis.

A question of primary concern to us is the treatment of glomus tumors. I have one case under observation at present whose symptoms began 15 years ago. This patient developed a paralysis and atrophy of one side of the tongue, paresis of the XI nerve, paresis of one side of the pharynx and also a dead ear. The VI and VII nerves are also affected. This patient is a cripple because she cannot swallow properly and has poor articulation. There are extensive changes shown on x-ray. In 1954 she was treated by radiation therapy. I saw her recently and she looked better than she did three years ago. However, she is still a cripple. This case illustrates the slow rate of progression of these tumors.

On the other hand, I recall four cases that I have operated on, three within the last few months, in two of which it was possible to

save even the eardrum and ossicular chain by removing the bone of the posterior and inferior canal walls, and then lifting the eardrum out of its sulcus. The tumor had not become fixed to the eardrum and after removal the drum was replaced. These were cases where the tumor apparently arose from the promontory (Jacobson's nerve), whereas in other cases it may arise from the bulb. In such early cases surgery certainly seems to be the treatment of choice. One of these early cases was treated by x-ray before surgery but showed no change in appearance and no relief of tinnitus, so I removed the tumor. I operated on another patient about three years ago with complete facial paralysis and symptoms of about four years' duration. The slides of Dr. Bast reminded me of that case because every mastoid cell bled furiously at operation, so I am now sure there was tumor all through those mastoid cells. I saw that patient a year later and there was a nice result. The cavity was healed and facial function had partially returned. I have not seen the patient for a year and I doubt that he is cured after seeing these slides, but he was greatly improved.

My opinion is that when the ear is functioning and destruction is not so extensive as to preclude removal with reasonable safety, these cases should be treated by surgery.

Dr. Williams gave a paper at the Otological Society a few years ago reporting very good results from x-ray therapy in this type of tumor. My own opinion is that we ought to use x-ray in those advanced cases where operation is not likely to be feasible. In the less advanced cases, particularly those which arise from the promontory or in the middle ear, surgery is the treatment of choice. Granted that we may not be sure of a cure, but if there should be recurrence it is likely to be recognizable and can be more easily dealt with.

Principles of the Use of Steroids in Otolaryngology

RACHMIEL LEVINE, M.D.

(by invitation)

(Abstract)

I shall talk on the principles of the use of the steroids, and leave out otolaryngology, since I know practically nothing of the field.

The word "steroids" in this context refers only to a few substances out of the very large group designated as steroids. The steroids referred to all have an oxygen atom at carbon 11 and at carbon 17, and they are designated as the 11-17 oxysteroids. Those are the substances that are in use for the alleviation of symptoms and signs in the various branches of medicine.

The adrenal cortex secretes three types of steroids. One is the type similar to desoxycorticosterone, the main action of which is to retain water and salt when given to the organism in vivo. This group of steroids is of no particular value or interest in the field we are exploring tonight.

The second group are the androgens secreted by the adrenal of both sexes. These androgenic substances also do not have many of the properties of the steroids we are interested in.

The adrenal cortex in addition secretes substances of the third type, which are the 11-17 oxysteroids. There is absolutely no relation between the function of the adrenal cortex as an endocrine gland and the use of the steroids in medicine. The use of these steroids is purely pharmacological, and the analogy which comes to mind right now is the use of insulin shock therapy in schizophrenia. Insulin shock therapy is not given because of an insulin deficiency. None of the disorders commonly treated with steroids, except for the diseases of the adrenal itself, have any relation to the function of the adrenal cortex. There is really no logical scientific basis for the widspread use of the steroids in medicine at all. If an endocrinologist had been asked in April, 1949, whether, on the basis of his knowledge of the function and physiological effects of the 11-17 steroids, he could deduce their eventual use in any nonadrenal disease, he would never have predicted their use as anti-inflammatory and anti-allergic agents.

We do have, however, a certain body of knowledge that has been gathered since May, 1949, as a result of the empirical alleviation of the symptoms of rheumatoid arthritis, which now gives us a background and a set of guiding principles as to the use of steroids in disease. We have also gathered a certain amount of knowledge about the structure of these compounds in relation to their therapeutic effects. Modifications from the original cortisone and hydrocortisone have appeared and been found helpful.

Soon after the discovery of Hench and his associates, the use of these compounds was extended to disorders which resembled rheu-

matoid arthritis or rheumatic fever, the collagen diseases. From this vantage point the use spread to the allergic disorders. Experimental observations demonstrated that a common feature of the action of cortisone was its anti-inflammatory effect. It suppressed granulation tissue proliferation in a variety of induced inflammations. This action is exerted locally in the inflamed site since one can inject a small amount of cortisone with the irritant into the same area, and inhibition of inflammation will occur. In general, one only has to use a very small amount of the substance when injected directly into the injured area without affecting the animal as a whole. Parallel with these animal investigations were the observations in the human. Surface inflammations such as conjunctivitis could be treated locally; so could single rheumatoid joints. It is apparent that the effect of cortisone is exerted directly in the area of inflammatory reaction. Whatever be the inflammatory stimulus, the normal reaction proceeds in a spherical manner from the site of the irritation. If cortisone is also present, the cells cannot react to the irritant. This was demonstrated in 1951 and 1952 by many experiments, of which the following is an example. It is known that one can produce experimental nephritis by injecting antibodies in an especially prepared animal. The antigen-antibody combination occurs in the basement membrane of the glomerulus and it serves as an inflammatory stimulus to the tissue around it. If the animal is pretreated with cortisone, the course of the disease is altered.

There is no evidence in the urine, no evidence in the behavior of the animal, and no evidence on pathologic section of the development of glomerulonephritis. When cortisone administration is interrupted, the glomerulonephritis develops as if the antibody were given at this time. As soon as the protective effect was removed, the inflammation occurred.

In rheumatoid arthritis, when steroid medication is stopped, flare-up of the rheumatoid process may occur. The steroids are in no sense curative.

The steroids are anti-inflammatory; their effect does not seem to depend on the nature of the inflammatory agent; they act locally; they do not represent anti-etiologic therapy; they simply relieve symptoms and signs associated with a simple or complex allergic or inflammatory reaction.

The steroids have, of course, actions other than anti-inflammatory. These have wrongly been called side-effects. They are really the exaggerated hormonal activities of these substances. Protein

breakdown is hastened, which on long-standing administration may lead to dangerous osteoporosis with fractures, especially of the vertebral column. The formation of sugar from protein is increased, and this may predispose, in about two per cent of the cases, to hyperglycemia. Sodium and water are retained, and there is an increase in the excretion of potassium. Thereby hypertension and edema may be produced. This skin thins and a redistribution of the fat occurs. This is the induced Cushing's syndrome seen formerly only in adrenal hyperplasia or tumor. These effects are inevitable whenever cortisone or one of its cousins has to be used in large dosage for long periods of time, such as in severe cases of lupus, pemphigus, periarteritis and in some cases of very severe inflammatory rheumatism. Below certain dosage levels these complications need not occur (usually below 50 mgm of cortisone or its equivalent). Another serious complication is of special interest to surgeons. Whenever any of the active steroids are administered to an individual, they raise the level of these materials in the blood. This serves to depress ACTH secretion by the pituitary. The patient's adrenals begin to diminish in size, and may become atrophic. Fortunately, in the overwhelming majority of instances, the process is reversible as soon as the administration of these drugs is stopped. However, when an individual has been under the influence of outside cortisone for too long, one should not remove medication abruptly, because the atrophic state of the adrenals may lead to an Addisonian crisis. When an additional strain is imposed on the individual, he may die in peripheral vascular collapse of the adrenal variety. One can overcome this by withdrawing the drug very gradually. This gives the pituitary a chance to increase its own activity and in turn bring the adrenals back to normal. There have been publications which can be interpreted to mean that in some instances such adrenal depressions are not reversible. I have not had that experience.

One of the ways to obviate the above difficulty is to give the cortical hormone by mouth every day and then periodically (every week or every two or three weeks) to inject ACTH. There is another way of obviating difficulty. It is possible sometimes to gain access to inflammatory or allergic processes without having to give parenteral medication. In dermatology, in otolaryngology, and certain other fields, the local application of the potent cortical hormones is being utilized, which has an excellent rational basis.

Now something about the substances themselves. Cortisone and hydrocortisone were the first compounds used. The ratio of activity,

if cortisone is 100 per cent, is 125 for hydrocortisone or about 25 per cent more potent per milligram. Of these, hydrocortisone is a normal hormone of the adrenal cortex. Meticortin and meticortilone are from four to five times as potent as the standard, per milligram. That by itself has no advantage; it does not matter at all whether something is more potent per milligram. The number of complications is about the same, but there is one advantage of meticortin and meticortilone, and that is that the retention of sodium and water is much less in therapeutic doses than it is with cortisone or hydrocortisone. There is a clinical suspicion that bleeding peptic ulcers are more common with the meticortins than with the original cortisone and hydrocortisone.

While the steroids prevent inflammatory reaction and pain, they, at the same time, prevent the formation of the barrier around the inflammatory focus. Therefore, viral and bacterial spread may occur. In the presence of any focus of infection the antibiotics have to be used simultaneously with the steroids. This same type of situation is probably the cause of the bleeding peptic ulcer. Peptic ulcer is an inflammatory nidus and has a barrier, but when steroids are given, two things happen. First, the acid-secreting activity of the stomach is enhanced, and, secondly, the healing process will be inhibited.

The steroids have been in some instances life-saving; in many more instances they have prevented pain and disability. However, they have also produced complications which we had not foreseen. The cortical substances are useful if intelligently handled, but they should not be dispensed as if they were harmless analgesics.

DISCUSSION

DR. FRANCIS L. LEDERER: I am sure that Dr. Levine has not made many friends tonight because he has failed to tell you that this is the "cure-all" to the many ills that befall our otolaryngological mankind. We are aware that since observations were made on the empirical bases referred to, there has been a good deal of abuse of their application. I suppose the next paper may be on the abuse of the steroids in otolaryngology. Each time we do not know what to do next, and when aspirin or antibiotics have not come to the rescue of the patient, we now use steroids. The detail salesman is our best bet.

Today we are residing in a generation that when we do not know something, we give cortisone. In desperation we have been using these preparations in the treatment of nasal polyps. We have been over-

whelmed and delighted in that we found that polyps presenting themselves in the anterior nares would disappear. The patient, of course, was so grateful he wanted to keep on taking these agents and did not want them discontinued, particularly when, if they were discontinued, the polyps recurred. I am glad to be enlightened about the use of the steroids and the terrible "side-effects" which now must be called complications. We have in the past been faced with disagreeable complications, particularly infections, because there was no resistance on the part of the body structure. Tonight we see just why this has occurred.

Now, surely, after listening to this comprehensive presentation, we will hesitate before we prescribe this type of remedial agent.

I was interested in the fact that in his early remarks, Dr. Levine mentioned that the emotions had something to do with the way these agents acted. We formerly did hear of patients who were given this remedy for rheumatoid arthritis, for example, who would become so euphoric as to practically be climbing the ceiling. I want to ask Dr. Levine whether this is still to be reckoned with as a complication, or whether this has been overcome.

DR. PHILLIP MOZER: Dr. Levine, I wish to ask what you feel are the prospects for the topical application of the cortisone substances. Is there absorption after prolonged use?

DR. RACHMIEL LEVINE: It depends on the amount and type you use. Fluorohydrocortisone is a very potent agent. It is practically impossible to give it parenterally since it is a powerful sodium retainer. Topically it is quite useful. One of the advantages of fluorohydrocortisone is that one can gauge the amount absorbed and keep it under the amount which will produce any systemic symptoms. Usually cortisone, by whatever route, is absorbed. One of the interesting things is that it is usually metabolized or excreted in 12 to 24 hours so that there is no cumulative effect. One might give 100 mg of hydrocortisone, and suppose this were all retained for a day and not excreted or metabolized but distributed throughout the total body water. In an adult the total body water is about 50 liters, so the concentration would be 100 mg in 50 liters, or 100 in 50,000 milliliters, or 1/5 of a mg per 100 cc. If one puts on topically ½ mg on the particular area, you are getting probably ten times the amount you actually need. That is why it is better if you can get a little bit where you want it. In a systemic disease there is the

difference that one cannot apply a little bit over the lesions of lupus, for instance.

Dr. Lederer mentioned the complication of psychosis from therapy with steroids. This is not as threatening or the damage as great as we used to think it would be, during the first six months of 1949 or in 1950. It occurs with much less frequency than I would have expected. On the other hand, peptic ulcer became apparent about 1950, and hemorrhage from this is now a more frequent complication.

DR. GAIL SOPER: What would be the care in cases you are going to do tonsillectomies on, if they have been on cortisone for more than two weeks and are still on it at present?

DR. LEVINE: Do you mean the patient is getting cortisone at the time of surgery?

DR. SOPER: Yes.

DR. LEVINE: If the patient is getting cortisone, continue it. The normal adrenal puts out in 24 hours somewhere between 12 and 24 mg of cortisone. All one needs, in the complete absence of adrenal function, is such an amount. Under stress, the normal adrenal secretes five times this amount. If anyone, therefore, is receiving something between 60 and 120 mg of cortisone or the equivalent for some systemic disorder and is being operated on, there is no need for additional cortisone. This person has as much as the normal adrenal will be able to put out under the most stressful conditions. If, however, the individual has had cortisone which has been discontinued and two weeks later he has to undergo operation, then one should give 100 mg of cortisone or the equivalent daily for several days, and then decrease it slowly.

DR. LEDERER: What about the factor of surgery and infection under those same circumstances?

DR. LEVINE: Infection is always a hazard. Whenever you fear infection, you have to use some of the broad-spectrum antibiotics. If you have a tuberculous patient particularly, there are the more specific antibiotics. Sometimes cortisone is used in infections. For example, in tuberculous meningitis in which there are many fibrous adhesions, the antibiotics may not be able to get to them too effectively, and cortisone will inhibit the formation of fibrous adhesions

and may lead to lysis. Therefore, cortisone is given with a high level of streptomycin and other antituberculous agents, in order to overcome such infections.

DR. ARTHUR RATKO: Does the topical application of these substances have an effect on the healing process?

DR. LEVINE: On the healing of wounds?

DR. RATKO: Yes. I wondered about the healing of wounds by first intention. Is there any explanation for the different action in wound healing? Can you explain why the normal expected response does not take place? The healing process, after all, is merely a modified form of an inflammatory process. Why should there be a differance between the alteration of the inflammatory response and the apparent failure to affect healing?

DR. LEVINE: I do not know. It is quite possible that if you took cortisone or fluorohydrocortisone and put it on the edges of the wound it might interfere with its healing, but I doubt that the concentration it gets would be high enough to interfere with the normal stimulus to repair. It seems to be a somewhat different process, for example, than in inflammation of infection.

DR. BURTON SOBOROFF: Is there any evidence to show that the final fibrosis and scarring is a chemical reaction, and does this make it any less, more, or just as likely with the use of the cortisone substances?

DR. LEVINE: This is very difficult to state because it is not really known. Cortisone has been used, for example, in keloids. Some have reported benefit; other workers have not seen any change. Cortisone has been given to prevent adhesions in the peritoneal cavity to aid in certain aspects of gastrointestinal surgery, but in these areas it is still open to investigation, and there are no definite data available.

DR. ARTHUR LOEWY: The steroid substances for topical use are available in strengths of from 0.5 to 2.5 per cent. Is there a proportionate difference in their local action, or is there a maximum topical strength beyond which any increase is not any more clinically effective?

DR. LEVINE: I think that would depend on the extent of the reaction and how near you can get to it with the material. This is

purely speculation, because I do not think it is known. There may be an error of 100 per cent up or down.

DR. ROBERT HENNER: Why has there been sensitivity to the mycins when used in combination with cortisone? Is there a technical difference in the sensitivity reaction to the mycins?

DR. LEVINE: There are certain inflammatory and allergic reactions where cortisone will have an effect. In the anaphylactoid reactions cortisone does well. On the other hand, in the Arthus phenomenon the ulceration does not seem to be prevented. So there is a difference between these inflammatory reactions.

DR. JOHN R. LINDSAY: In fibrous tissue formation in case of burns of the esophagus, is there any rationale to the use of cortisone?

DR. LEVINE: Yes, there is some rationale to that. I would say if steroids are given very early, immediately after the burn, during the time there is serous exudation and edema, there might be benefit. I doubt very much if cortisone would help after the fibrous tissue had formed.

DR. LINDSAY: Could local applications be used, or would parenteral administration be best?

DR. LEVINE: It would seem that you would have to use large parenteral doses because the local reaction would be a diffuse one.

Books Received

The Year in Otorhinolaryngology: 1957. (L'Année Oto-rhino-laryngologique: 1957.)

Lectures in otorhinolaryngology at the Hôpital Lariboisiere and la Pitié. Paper, 8vo, 224 pp., 112 illustrations. Paris, Masson et Cie and Librarie Arnette, (In French). Price, 1,500 francs.

Tinnitus. (Les Bourdonnements d'Oreille.)

By *A. Appaix, J. Bouche* and *G. Bremond.* Communication to the Congress of the Société Française d'Otorhinolaryngologie, 1957. Paper, 8vo, 240 pp., 31 illustrations. Paris Librarie Arnette (In French). Price, 2,000 francs.

Malignant Tumors of the Ear (Exclusive of the Internal Ear). (Les Tumeurs Maligne de l'oreille.)

By *J. Leroux-Robert* and *A. Enneyer.* Communication to the Congress of the Société Française de Otorhinolaryngologie, 1957. Paper, 8vo, 336 pp., 122 illustrations. Paris, Librarie Arnette. (In French). Price, 3,000 francs.

Ear, Nose and Throat Dysfunctions Due to Deficiencies and Imbalances

By Sam E. Roberts, M.D., Associate Professor of Otolaryngology, University of Kansas School of Medicine, with a foreword by Morris Fishbein, M.D. Cloth, xxiii - 323, Charles C. Thomas, Publisher, Springfield, Ill., 1957 (Price $8.50).

For a long time Doctor Roberts has been thinking and writing about ear, nose and throat troubles in the light of disruptions elsewhere in the body—nutritional, glandular, psychosomatic—and has now gathered his conclusions into a very provocative book.

This book is probably best characterized by Dr. Roberts himself in the opening sentence of his introduction: "It is forthwith disclosed that the opinions and annotations in this book are founded on clinical observations, and the results described are, in part, subjective." For deficiencies and imbalances there are no laboratory tests, he affirms, and whether one goes along with that or not, there is still much to be said for a good detached, if sometimes unscientific, look at a sick man instead of merely a man's sickness; also for using one's own head instead of someone else's statistics.

Lymphatics, Lymph and Lymphoid Tissue

By Joseph Mendel Yoffey, D.Sc., M.D., Professor of Anatomy, University of Bristol, and Frederick Colin Courtice, M.A., D.Phil., D.Sc., F.R.A.C.S., Director Kanematsu Memorial Institute of Pathology, Sydney Hospital, Sydney. Cloth, 8vo., vi - 510 pp., illustrated, Harvard University Press, Cambridge, Mass., 1956 (Price $10.00).

This is an extensive revision of the classical volume of the same name by Drinker and Yoffey, F. C. Courtice now replacing the late C. K. Drinker as co-author.

Besides the structure and functions of the lymphatic system and its disposal in various regions and tissues, the authors discuss such things as capillaries and capillary permeability, extravascular circulation of protein and related subjects closely connected with nasal diseases, and those of the throat and neck.

Yoffey's contributions, extending over a number of years, on the absorption of bacteria, dyes, particulate matter and a variety of drugs have added materially to our knowledge of upper respiratory infections and their treatment.

Headache, Diagnosis and Treatment

By Robert E. Ryan, B.S., M.D., M.S. (in Otolaryngology), F.A.C.S., Department of Otolaryngology, St. Louis University School of Medicine, Associate Otolaryngologist, St. John's Hospital; Assistant Otolaryngologist, St. Louis University Group of Hospitals, St. Louis, Mo. Cloth, 421 pp., C. V. Mosby Company, St. Louis, Mo., 1957 (Price $6.75).

This is the second edition of a monograph which first appeared three years ago. It is written in textbook form with frequent topical headings for easy reference and study. The first eight chapters are devoted to the mechanics and underlying causes of head pain and the theories of treatment, the remainder to some forty specific types.

Regulation and Mode of Action of Thyroid Hormones

Editors for the Ciba Foundation, G. E. W. Wolstenholme, O.B.E., M.A., M.B., B.Ch., and Elaine C. P. Millar, A.H.-W.C., A.R.I.C. Cloth, xii - 311 pp., Little, Brown and Company, Boston, Mass., 1957 (Price $8.50).

Eighteen papers, and discussions, constituting one of a series of colloquia on endocrinology.

Ortho- and Pathobiology of the Labyrinth (Die Ortho- und Pathobiologie Des Labyrinthes)

By Karl Wittmaack, Professor and Director of the University Ear, Nose and Throat Clinic, Hamburg, and Heinz Rollin. Cloth, 8vo., xi - 294 pp., illustrated, Georg Thieme, Stuttgart, Germany, 1956 (Price $8.60). In U.S.A. and Canada Inter-Continental Medical Book Corp., New York.

Exhaustive treatise under three headings: I. Orthobiology of the Inner Ear; II. Pathobiology of the Inner Ear; III. Clinical Consideration of Dystonic and Dystrophic Disease Processes of the Inner Ear and the Auditory Nerve.

Symptoms and Diagnosis of Ear, Nose and Throat Diseases (Symptome und Diagnose Der Hals-Nasen-Ohren-Krankheiten)

By Prof. Dr. M. Schwarz. Pp.: vi - 351, illustrated, Georg Thieme Verlag, Stuttgart. In the United States Intercontinental Medical Book Corporation, New York, 1956 (Price $11.40).

A comprehensive monograph, presented with the usual German attention to detail. Well illustrated. No documentation.

The Early Diagnosis and Treatment of Acoustic Nerve Tumors

By J. Lawrence Pool, M.D., Professor of Neurological Surgery, Columbia University, Director of Service of Neurological Surgery, The Neurological Institute, Presbyterian Hospital, New York, and Arthur A. Pava, M.D., Assistant Neurological Surgeon, Wesson Memorial Hospital, Springfield, Mass. Cloth, 161 pp., illustrated, one color plate, Charles C. Thomas, Publisher, Springfield, Ill., 1957 (Price $5.50).

A summary of observations based upon a series of 135 cases.

Hormones in Blood

Editors for the Ciba Foundation, G. E. W. Wolstenholme, O.B.E., M.A., M.B., B.Ch., and Elaine C. P. Millar, A.H.-W.C., A.R.I.C. Cloth, x - 416 pp., Little, Brown and Company, Boston, Mass., 1957 (Price $9.00).

Twenty-one papers comprising a colloquium, dealing chiefly with transportation of hormones in the blood and the measurement of hormone levels in the blood.

Noise and Your Ear

By Aram Glorig, Jr., Director of Research, Research Center of the Subcommittee on Noise in Industry, Committee on Conservation of Hearing of the American Academy of Ophthalmology and Otolaryngology, Los Angeles, California. Cloth, viii - 152 pp., illustrated, Grune and Stratton, New York and London, 1958 (Price $6.50).

"This monograph is a survey of existing information about hearing loss and noise . . . written primarily for persons who require only a general knowledge of the subject . . ."—*from the Preface.*

The Vestibular Apparatus (L'Appareil Vestibulaire)

By A. Aubin, Professor of Clinical Otorhinology of the Faculty of Medicine of Paris. Paper, 603 pp., illustrated, University Press of France, Publisher, Paris, France, 1957.

Neuropathy of the VIII Nerve (Neuropatie dell' VIII)

By G. Borri and D. Felisati, Otorhinolaryngological Clinic of the University of Milan. Paper, 65 pp., Supplement XXXI of the Italian Archives of Otology, Rhinology and Laryngology.

Diseases of the Internal Ear and Oto-Neurology (Maladies de L'Oreille Interne et Oto-Neurologie)

By M. Aubry, Professor of the Faculty of Medicine in Otorhinolaryngology of the Hospital of Paris, and P. Pialoux, Otolaryngologist of the Hospital of Paris. Cloth, vii - 706 pp., illustrated, in French, Masson et Cie, Publisher, Paris, France, 1957.

A comprehensive text, well presented, and for the French-reading student an easy reference book. Notable diagrams and illustrations.

Oral Medicine

By Lester W. Burket, A.B., D.D.S., M.D., Sc.D., Professor of Oral Medicine and Dean, School of Dentistry, University of Pennsylvania; Professor of Oral Medicine, Graduate School of Medicine, University of Pennsylvania; Honorary Professor, National University, Bogotá, Colombia; Chief, Oral Medicine Service, Philadelphia General Hospital; Consultant to the Army, Navy and Veterans Administration. Cloth, xxi - 558 pp., illustrated, Third Edition, J. B. Lippincott Company, Philadelphia, Pa. (Price $14.00).

The Palatine Tonsil and Immunity (Tonsille Palatine e Immunità)

By G. Borasi, M.D., and D. Canciullo, M.D. Paper, 216 pp., illustrated, Luigi Parma, Bologna, Italy, 1956.

Peristalsis and Antiperistalsis in the Esophagus (Peristalsi ed Antiperistalsi dell' Esofago)

By Prof. Antonino Clementi. Cloth, 93 pp., Cappelli Editore, Bologna, Italy, 1956. (In Italian)

Speech Audiometry (Sprachaudiometrie. Grundlagen und Praktische Anwendung Einer Sprachaudiometrie für das Deutsche Sprachgebiet)

By Doz. Dr. Med. Karl-Heinz Hahlbrock, Freiburg. Paper, vi - 182 pp., illustrated, Georg Thieme, Stuttgart, Germany, 1957 (Price $5.00). In U.S.A. and Canada Intercontinental Medical Book Corp., New York.

Anatomic Nomenclature (Nomina Anatomica)

By Prof. Dr. Fr. Kopsch. Paper, 155 pp., Georg Thieme, Stuttgart, Germany, 1957 (Price $1.55).

A comparative review of the Basel, Jena and Paris nomenclatures arranged in tabular form.

Notices

THE AMERICAN OTOLOGICAL SOCIETY

The American Otological Society will hold its ninety-first meeting May 17-18, 1958, at Hotel Fairmont, San Francisco, California.

PROGRAM

Effect of Tractor Noise on Hearing of Farmers

Dean M. Lierle, M.D.

Scott N. Reger, Ph.D.

Anatomical Structure of the Stapes and the Relation of the Stapedial Footplate to Vital Parts of the Otic Labyrinth

Barry J. Anson, Ph.D.

Theodore Bast, Ph.D.

Repair and Consequences of Trauma to the Stapes and Oval Window in Experimental Animals

Richard J. Bellucci, M.D. (by invitation)

Dorothy Wolff, M.D. (by invitation)

Further Studies on the Function of the Utricular Macula

J. A. Sullivan, M.D.

Walter Johnson, M.D. (by invitation)

Brydon Smith, M.D. (by invitation)

Hydro-Dynamic Origin of Aural Harmonics in the Cochlea

Juergen Tonndorf, M.D. (by invitation)

Destructive Labyrinthotomy—A Study of the Prognosis of Postoperative Disability

K. M. Simonton, M.D.

P. A. Sciarra, M.D. (by invitation)

The Present Status of the Electrophysiology of the Cochlea

Hallowell Davis, M.D.

Functional Changes in Inner Ear Deafness

Merle Lawrence, Ph.D. (by invitation)

ADDRESS OF THE GUEST OF HONOR

Ralph Fenton, M.D.

Observations on Temporary Threshold Shift Resulting from Noise Exposure

Aram Glorig, M.D.

Differential Diagnosis Between Otosclerosis and Congenital Footplate Fixation

Howard P. House, M.D.

Discussion: Dorothy Wolff, Ph.D. (by invitation)

The Technique for Removal and Preparation of Temporal Bones for Anatomical Surgical Dissection and Pathologic Study

Harold F. Schuknecht, M.D.

Studies Concerned with Tubo-tympanitis

Ben Senturia, M.D.

Discussion: Gordon D. Hoople, M.D.

Neurological Assessment of Some Deaf and Aphasic Children

R. Goldstein, Ph.D. (by invitation)

W. M. Landau, M.D. (by invitation)

F. R. Kleffner, Ph.D. (by invitation)

Hallowell Davis, M.D.

S. Richard Silverman, Ph.D.

The Potential Value of Pressures of the Labyrinthine Fluids as Measured with an Electromanometer

Francis L. Weille, M.D.

John Irvin, M.D. (by invitation)

Discussion: Nelson Kiang, M.D. (by invitation)

The Differential Diagnosis of Ménière's Disease

Henry L. Williams, M.D.

Otosclerosis in Ten Pair of Identical Twins

Edmund P. Fowler, M.D.

The profession is cordially invited to attend.

THIRD INTERNATIONAL CONGRESS OF ALLERGY

The Third International Congress of Allergy will take place in Paris, France, October 19-26, 1958, sponsored by the International Association of Allergology and the French Allergy Association.

There will be a symposium on asthma and emphysema and related subjects besides sectional papers on allergy. Convention tours and a very enjoyable social program have been arranged.

For all information please write to: Dr. Samuel M. Feinberg, 303 East Chicago Avenue, Chicago, Illinois, U.S.A.; Dr. José M. Quintero Fossas, Paseo 313, Vedado, Havana, Cuba; or Dr. Bernard N. Halpern, 197 Boulevard St. Germain, Paris 7, France.

CALIFORNIA MEDICAL ASSOCIATION

The Ear, Nose and Throat Section of the California Medical Association has arranged an interesting scientific program for its next meeting on Monday, April 28, 1957, 9 A.M., at the Ambassador Hotel in Los Angeles, consisting of the following papers:

The Endonasal Approach to Nasopharyngeal Tumors, by Joel J. Pressman, M.D., Los Angeles.

Changing Aspects of Surgery to the Middle Ear and Mastoid, by O. E. Hallberg, M.D., Rochester, Minnesota.

Interpretation of Dizziness Related to the Labyrinth, by Jack L. Mahoney, M.D., Monterey, California.

Aviation and the Otolaryngologist, by Bruce V. Leamer, M.D., Los Angeles.

Pathology and Therapy of the Singing Voice, by Paul J. Moses, M.D., San Francisco.

Delayed Skin Grafting of the Mastoid Cavity, by William F. House, M.D., Los Angeles.

THE GOULD AWARD

The Gould Award of $250, offered by the William and Harriet Gould Foundation of Chicago, Illinois, will be presented annually for outstanding research in Laryngology, to encourage and reward fundamental investigations in this field. While the award recognizes the completion of a specific research project, favorable attention will be given to investigators with a long-term interest in this subject.

The Award Committee is comprised of Sir Victor Negus, England; Prof. Cotohi Satta, Japan; Prof. Dr. Eelco Huizinga, The Netherlands; and the undersigned.

Hans von Leden, M.D., Medical Director

30 North Michigan Avenue

Chicago 2, Illinois, U.S.A.

UNIVERSITY OF TEXAS

The University of Texas Postgraduate School of Medicine and the Houston Speech and Hearing Center will present the second training course in The Rehabilitation of the Laryngectomized, June 9 - 20, 1958.

The course this year will cover Vocational Adjustment, Anatomy of the Speech Mechanism, Normal Speech and Voice Production, Esophageal Speech and Voice, Laryngeal Surgery, Radiation Therapy, Facts about Cancer and Techniques in Teaching Esophageal Speech, and will combine clinical and didactic approaches.

Applications for Traineeships, which are limited in number, should be made as soon as possible, and will not be considered after May 19, 1958.

Inquiries concerning this course should be directed to:

The University of Texas
Postgraduate School of Medicine
410 Jesse Jones Library Building
Texas Medical Center
Houston 25, Texas

SOCIETE FRANCAISE d'OTO-RHINO LARYNGOLOGIE

The Societe Francaise d'Oto-Rhino-Laryngologie will hold its annual congress in the grand Amphithéâtre de la Faculté de Médecine de Paris, rue de l'Ecole de Médecine, October 20-23, 1958.

The official topics for discussion will be:

1) Traitement du Cancer de las base de la langue.
Mm. Huet, Gignoux, Berard, Andre et Labayle.

2) La Cortisone et l'A.C.T.H. en O.R.L.
Mm. Aubin, Terracol et Guerrier.

For information please address the Secretary General, Dr. H. Guillon, 6, avenue Mac-Mahon, Paris.

ACADEMY SUBCOMMITTEE ON HEARING IN CHILDREN

The American Academy of Ophthalmology and Otolaryngology, through its Subcommittee on Hearing in Children of the Committee on Conservation of Hearing, has been conducting a long-term nationwide study of problems relating to the conservation of hearing in children. The specific aims are to develop the most efficient case-finding methods and to use these methods in estimating the magnitude of the problem in the country; to study state laws and review current practices and facilities for rehabilitation of hearing impaired children; to help develop methods for medical and surgical rehabilitation standards; and ultimately to use the Subcommittee findings in assisting professional workers to improve and enhance programs in hearing loss.

In the second year of operations, a full-time Executive Director, Eldon L. Eagles, M.D., has been engaged, and offices established at the Graduate School of Public Health, University of Pittsburgh. An initial study is being conducted in Pittsburgh to identify early medical signs and symptoms which may indicate danger of hearing impairment, to measure the psychological, social and other effects of such impairment and to develop efficient and economical methods for the testing of hearing in children. The Pittsburgh study is a cooperative effort among the following: the Subcommittee on Hearing in Children, the Graduate School of Public Health and the School of Medicine of the University of Pittsburgh, the Pittsburgh Board of Public Education, and the Allegheny County Department of Health.

The members of the Subcommittee on Hearing in Children are: Dr. John E. Bordley, Baltimore; Dr. Victor Goodhill, Los Angeles; Dr. Hollie E. McHugh, Montreal; Dr. S. Richard Silverman, St. Louis; and Dr. Raymond E. Jordan (Chairman), Pittsburgh. An advisory committee of consultants from the University of Pittsburgh includes Dr. Samuel M. Wishik, Dr. Leo G. Doerfler, and Dr. Isidore Altman. Grants from the United States Children's Bureau through the Pennsylvania Department of Health and from the National Institutes of Health are providing financial support.

MOUNT SINAI HOSPITAL

An intensive postgraduate course in Rhinoplasty, Reconstructive Surgery of the Nasal Septum and Otoplasty will be given July 12,

1958, to July 26; 1958, by Dr. Irving B. Goldman and staff at the Mount Sinai Hospital, New York, in affiliation with Columbia University.

Candidates for the course should apply to the Registrar for Postgraduate Medical Instruction, the Mount Sinai Hospital, Fifth Avenue and One-hundredth Street, New York 29, New York.

DEUTSCHE GESELLSCHAFT DER HALS-, NASEN-, OHRENÄRZTE

The Deutsche Gesellschaft der Hals-, Nasen-, Ohrenärzte will hold its annual meeting in Salzburg, Austria, May 19-22, 1958.

The official general topics for discussion will be related to the nasal mucosa, and recent discoveries pertaining to it.

Prof. Dr. Hofer, *President*; Prof. Dr. H. Leicher, Mainz, *Secretary*

SURGERY OF DEAFNESS

A course in the Surgery of Deafness will be given under the direction of Prof. A. Aubin, June 9-14, 1958, at the Hopital Lariboisière, Paris. Address: Cours de Technique Cirurgicale, Servide d'Otorhinolaryngologie, Pavillon Isambert, Hopital Lariboisière, 2 Rue Amboise-Paré, Paris X, France.

SEVENTH INTERNATIONAL CONGRESS OF BRONCHOESOPHAGOLOGY

The Seventh International Congress of Bronchoesophagology will take place September 12 - 14, 1958, at Kyoto, Japan. The summary of papers, within 800 words, should be sent to the director not later than the end of April, 1958.

Director: Prof. Mituharu Goto, M.D.
Otorhinolaryngological Clinic
Kyoto University Hospital
Kyoto, Japan

THE BELGIAN SOCIETY OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY

The Belgian Society of Otology, Rhinology and Laryngology will hold an extraordinary congress at Brussels from June 2nd to 7th, 1958 under the patronage of the Commissariat General of the Belgian Government at the Universal and International Exhibition, Brussels, 1958.

For information, apply to the General Secretary, Dr. Henschel, C.E.L., 19, Rue de Fétinne, Liège, Belgium.

INDIANA UNIVERSITY

The 43rd Annual Postgraduate Course in Anatomical and Clinical Otolaryngology will be presented by the Indiana University School of Medicine, March 24 to April 5, 1958.

Primarily an intensive course in anatomy of the head and neck, this course includes dissection and devotes 16 hours to the histopathology of otolaryngology. Lectures and demonstrations are designed as a review of basic principles and of recent advances in otolaryngology and bronchoesophagology.

Limited to 24 registrants, the course is open to physicians specializing in ear, nose and throat (eye) and to residents in training in this specialty.

For information address: Division of Postgraduate Medical Education, Indiana University School of Medicine, Indianapolis 7, Indiana.

UNIVERSITY OF MIAMI

The Fourth Postgraduate Course in Esophageal Speech and Organic Voice Problems will be given by the University of Miami School of Medicine, June 16-27, 1958. Specialists in otolaryngology, physical medicine and rehabilitation and speech therapists are eligible. Government grants for training, valued at $250 each, for tuition and traveling expense are available on application. Address: Dr. Homer F. Marsh, Dean, University of Miami School of Medicine, Coral Gables, Florida.

AMERICAN LARYNGOLOGICAL ASSOCIATION

Copies of the Transactions of the American Laryngological Association are available for general distribution at $8.00 a copy. Please send request with check to Dr. Edwin N. Broyles, Editor, Transactions, 1100 North Charles St., Baltimore 1, Md.

AMERICAN SOCIETY OF FACIAL PLASTIC SURGERY

The American Society of Facial Plastic Surgery will hold its annual Spring Meeting on March 10 and 11, 1958, at Columbus, Ohio.

AMERICAN ASSOCIATION FOR CLEFT PALATE REHABILITATION

The American Association for Cleft Palate Rehabilitation will hold its 16th Annual Convention at the St. Francis Hotel in San Francisco on Thursday, Friday and Saturday, April 24, 25 and 26, 1958.

The association is composed of medical, dental and paramedical specialists who are interested in the rehabilitation of persons with cleft lips and palates.

ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY

The Annals Publishing Company will pay $2.00 each for copies of the ANNALS of March 1957, which is out of print.

Please address the Business Manager, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, P. O. Box 1345, Central Station, St. Louis 1, Mo., U.S.A.

UNIVERSITY OF MANCHESTER

An International Congress on the Educational Treatment of Deafness will be held at the University of Manchester, England, July 15th to 23rd, 1958, with the approval and support of the Council of the University, with financial help from the Ministry of Education, the National College of Teachers of the Deaf, and the Rathbone Trustees, and with the co-operation of the British Council and the Alexander Graham Bell Association for the Deaf, Inc., Washington, D.C., U.S.A.

Forms of application for membership of the Congress will be supplied on request to Professor A. W. G. Ewing, Department of Education of the Deaf, The University, Manchester 13, England.

OFFICERS

OF THE

NATIONAL AND INTERNATIONAL OTOLARYNGOLOGICAL SOCIETIES

American Academy of Ophthalmology and Otolaryngology

President: Dr. LeRoy A. Schall, Boston
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.

American Board of Otolaryngology

President: Dr. Gordon D. Hoople, Syracuse, N.Y.
Secretary: Dr. Dean M. Lierle, University Hospital, Iowa City, Iowa

American Broncho-Esophagological Association

President: Dr. Walter Hoover, Boston, Mass.
Secretary: Dr. F. Johnson Putney, 1719 Rittenhouse Sq., Philadelphia 3, Pa.
Meeting: San Francisco, Calif., May 21, 22, 23, 1958

American Laryngological Association

President: Dr. Harry P. Schenck, Philadelphia
Secretary: Dr. James H. Maxwell, University Hospital, Ann Arbor, Mich.
Meeting: San Francisco, May 19-20, 1958

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. Percy Ireland, Toronto, Canada
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester, N. Y.

American Medical Association, Section on Laryngology, Otology and Rhinology

Chairman: Alden Miller, M.D., Los Angeles
Secretary: Hugh A. Kuhn, M.D., Hammond, Ind.

American Otological Society

President: Dean M. Lierle, M.D., Iowa City
Secretary-Treasurer: Lawrence R. Boies, M.D., University of Minnesota Hospitals, Minneapolis 14, Minnesota

The American Society of Ophthalmologic and Otolaryngologic Allergy

President: Joseph W. Hampsey, M.D., Grant Bldg., Pittsburgh 19, Pa.
Secretary-Treasurer: Daniel S. DeStio, M.D., 121 S. Highland Ave., Pittsburgh 6, Pa.
Meeting: Palmer House, Chicago, Illinois, October 16 and 17, 1958

Pacific Coast Oto-Ophthalmological Society

President: H. Leroy Goss, M.D., 620 Cobb Building, Seattle 1, Wash.
Secretary-Treasurer: Homer E. Smith, M.D., 508 East South Temple, Salt Lake City, Utah

The Society of Military Otolaryngologists

President: Captain William C. Livingood, United States Navy (MC)
Secretary-Treasurer: Lt. Colonel Stanley H. Bear, United States Air Force (MC)

Canadian Otolaryngological Society

President: Dr. G.M.T. Hazen, 208 Canada Bldg., Saskatoon, Sask.
Secretary: Dr. G. A. Henry, Medical Arts Bldg., Suite 328, Toronto, Ont.
Meeting: Halifax, N.S., June 9-11, 1958

International Bronchoesophagological Society

President: Dr. Jo Ono, Tokyo
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40, Pa.
Meeting: Kyoto, Japan, Sept. 12-14, 1958

Pan-American Association of Oto-Rhino-Laryngology and Broncho-Esophagology

President: Dr. Jose Gros, Havana
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40, Pa.
Meeting: Sixth Panamerican Congress, Brazil, August 12, 1958